ARCHIVES of OPHTHALMOLOGY

VOLUME 7 (old series Volume 64) MAY, 1932 NUMBER 5

ELECTRO-ENDOTHERMY IN DETACHMENT OF THE RETINA

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The hopelessness that used to characterize the ophthalmologist's attitude toward the question of treatment for retinal detachment has to some extent begun to lessen. Prospects have become brighter with the operative method of treatment suggested by Gonin. There is no doubt that ophthalmologists stand in great debt to Gonin for having so successfully and energetically taken up this question. A task of this nature becomes the more exacting as it involves, and will always involve, many great disappointments for the operator. Thanks to Gonin's work, the operative treatment for retinal detachment has become one of the vital issues in ophthalmology.

Gonin's view of the pathogenesis of and operative treatment for detachment of the retina are already well known to every ophthalmologist. He maintains consistently de Wecker's old theory of the importance of a rupture (hole) in the retina, as the primary factor in the origin of the detachment. If the hole can be made to close, the subretinal contents are absorbed and the detached retina resumes its normal position. By direct cauterization of the margins of the hole, Gonin tries to bring about its closure.

In his own hands this method has yielded brilliant results. Equally successful results have not, as a rule, been obtained by his followers (table 1), even if the results as compared with those of methods employed earlier must be regarded as good.

The question may be raised as to the reasons for trying to replace Gonin's undoubtedly good method by something different. This seems to me justified for several reasons.

It cannot be said to have been proved beyond doubt that rupture (hole) is the immediate cause of detachment of the retina. It is true that holes are often observed in detachment of the retina, but cases do occur in which such holes cannot be detected in spite of the closest search. That the presence of a hole in the retina is of the greatest import for the persistence of the detachment and constitutes a bar to reattachment is a fact long known. But this need not mean that the hole is in every case, from an etiologic point of view, the primary or the main

Submitted for publication, Jan. 14, 1932.

factor. I suppose that a degenerated retina, presumably present in ablatio retinae, might rupture if distended by subretinal fluid.

This fluid may conceivably be formed by inflammatory exudate, by abnormal filtration because of vascular changes or by filtration through the vessel walls "ex vacuo" when the already frail connection between the pigment epithelium and the other layers of the retina for some reason has become still looser. The last-mentioned process may be

TABLE 1.—*Survey of Results Obtained by a Few Operators in Cases of Detachment of the Retina* *

Author	Method	Number Patients Examined	Number Patients Operated On	Number Patients Recovered	Number Showing Improvement	Recoveries, per Cent
Gonin (1929)	Gonin's operation	100	81	43	..	53
Arruga	Gonin's operation	...	33	?	?	?
Bruckner (1929)	Gonin's operation	...	12	5	..	42
Vogt (1930)	Gonin's operation	...	26	11	..	42
Meller (1930)	Gonin's operation	...	50	..	..	12
Lindner (1930)	Gonin's operation	42	33 (Whole material)	13	..	39
Amsler (1931)	Gonin's operation	74	29	12	..	41
Igersheimer (1931)	Gonin's operation	...	21	5	..	24
Bielschowsky (1931)	Gonin's operation	...	18	2	..	11
Fleischer (1931)	Gonin's operation	...	12	1	..	8
Clausen (1931)	Gonin's operation	...	30	...	..	25
Stein (1931)	Gonin's operation	55	41	10	..	24
Wessely (1931)	Gonin's operation	...	18	3	4	17 (+ about 22% improved)
Doggart and Shapland (1931)	Gonin's operation	...	75	24	12	32 (+ about 16% improved)
Gonin (1931)	Gonin's operation	300	221	118	..	53
Larsson (1932)	Electro-endothermy and trephining	...	40	20	..	50

* The table does not claim to be complete, but is meant only to illustrate the results obtained according to reports of the last two years. A great many brief reports have been published, comprising a smaller number of cases.

regarded as a sequel or an expression of the degenerative changes in the retina.

A hole may be considered as a secondary factor in relation to the detachment, but, once there, it constitutes a most formidable bar to spontaneous healing.

Obviously Gonin's operation cannot be utilized in cases of retinal detachment in which no hole can be found. Such cases are also excluded by Gonin himself from operative treatment.

Whether the rupture in the retina is of primary or secondary nature, it is clear that closure is of the greatest importance. At least Gonin formerly believed that by his method of operating he obtained closure

of the hole by causing union of the margins of the rupture. This mode of healing is, however, rather unlikely. It is difficult to imagine that the delicate retina would itself be capable of such a healing process. The fact that the detached retina is sometimes reattached after a galvanocauterization that does not involve the site of the hole is definitely against this view of the healing process, as is the fact that good results are obtained by electro-endothermy—to be described—although probably no great direct thermal effect is produced.

It seems to me that the closure of the hole and the favorable result of Gonin's operation are in all probability brought about by adhesion of the margins of the rupture to the underlying choroid, or perhaps through some cicatricial fixation to the choroid of some larger retinal area within which the hole is located.

Gonin asserts that if the hole is closed by operation, reattachment of the retina takes place. This thesis, however, can equally well be reversed: When reattachment of the retina takes place and the retina becomes adherent to the choroid, the hole heals or becomes obliterated. When Gonin fails in his operation, he blames the nonclosure of the hole, which remains in its original state. This thesis can also be reversed: The hole remains because the retinal detachment still persists and the retina is not in contact with the choroid.

I would emphasize, however, once more that closure of the hole or rather the prevention of communication between the preretinal and the subretinal spaces, when a hole or communication exists, must naturally be considered of the utmost importance for a favorable operative result. Great credit is due to Gonin for having drawn attention to this factor in his operation.

As ablatio retinae and holes are assumed to occur because of certain degenerative changes in the retina, especially in its peripheral part, or because of uncertain changes in the choroid, the object of treatment would be to attain an adherence to the underlying tissue along the whole peripheral area of the retina. On these grounds it seems to me desirable that in the operation an adhesion should be striven for between the retina and the choroid that will firmly fix the whole of that area of the retina which particularly seems to constitute a locus minoris resistentiae, in so far as its tendency toward detachment is concerned. This part may be assumed to be the area within which the hole is located or, when no hole is present, where the detachment comes first in evidence. Often, perhaps, this liability to detachment may be well marked in the whole peripheral area of the retina; such cases are perhaps prognostically the most unfavorable. In other cases the changes in the retina or choroid are perhaps confined to a smaller area. Such cases are more suitable for operative treatment.

Gonin's operation is restricted to a relatively small area. This probably also explains why the operation is of no use in cases of extensive or numerous holes and, likewise, why the operation has to be so often repeated. By the method of electro-endothermy that I have tried, an effect is obtained over a more widespread area.

I have set out these views in order to explain why I consider it theoretically justifiable to seek new methods for the operative treatment for retinal detachment.

Technically, Gonin's operation is not easy on account of the difficulty in localizing the hole exactly. The relatively small operative field renders the exact localization of the hole extremely important.

My aim has been to establish a wide adherence of the detached retina to the underlying tissue. This goal I have tried to reach by making use of electro-endothermy transclerally, obtaining a deep thermal effect without necrotization of the sclera.

In 1923, I carried out some experimental tests with this method and found the sclera of the rabbit's eye to be very resistant. By applying the treatment to the sclera a deep thermal effect was obtained that led to extensive changes of the fundus and opacities of the vitreous humor without causing necrosis of the sclera. At that time the experiments were confined to animals. After I had become more familiar with electro-endothermy as a clinical therapeutic method for the removal of epibulbar tumors,[1] and after Gonin's method and results had become better known, electro-endothermy, as originally intended, came to be clinically used also in cases of retinal detachment.

The method of operation may be described thus:[2]

The conjunctiva is detached at the corneoscleral boundary and stripped off over an area corresponding to the extent of the retinal detachment. The globe is carefully exposed. In order to gain complete access to the globe a canthotomy is, as a rule, carried out and, if necessary, the tendinous attachments of one or more muscles are divided. The conjunctiva is kept away from the operative field by sutures held and fixed by artery clamps, so placed that the conjunctiva is thereby held expanded and the sclera kept free. In some cases it is better to fix the sutures further back in the fascia tenoni as the conjunctiva may easily be torn. By sutures or hooks in the muscular attachments and by small Jäger's plates, especially designed for the purpose of pushing aside fascia tenoni and the orbital contents, the globe is made accessible to the operator. After careful arrest of bleeding, best done by electro-endothermy (with a needle-shaped active electrode), the endothermal treatment proper is begun.

The large indifferent electrode made up of a lead plate is applied to the leg or thigh; to obtain good contact the lead plate is not fixed directly on the skin, but

1. Larsson, S.: Surgical Diathermy in Certain Diseased Conditions of the Eye, Acta ophth. **3**:319, 1926; Electroendothermy in Ocular Leprosy, ibid. **6**:344, 1928.

2. This was first described by me in 1930 (Larsson, S.: Acta ophth. **8**:172, 1930).

an intervening gauze pad soaked in common salt solution is applied. The lead plate is bound to the leg by means of a gauze bandage, so as to obtain complete contact over a large surface area. This is important to prevent burns. As the active electrode a metallic ball, 0.66 mm. in diameter and provided with a handle, is used.

An extremely weak current is used; on contact between the active electrode and the terminal of the indifferent electrode, the amperemeter of the apparatus generally gives a reading of from 1 to 1½ amperes. On contact between the active electrode and the sclera the current is too weak to give any reading on the amperemeter (less than 100 ma.). At several places, a few millimeters apart, the active electrode is brought in full contact with the exposed sclera for five seconds at the most. To avoid contact with the ciliary body, the treatment is carried out at a respectful distance from the limbus, from 8 to 9 mm. This is done in order not to risk the production of cataract or other complications that may conceivably arise from damage to the ciliary body. The thermal effect on the sclera is evident as an opaque, generally dark-colored, annular zone corresponding to the place of con-

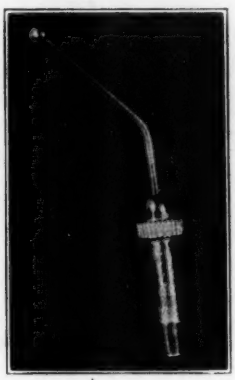

Fig. 1.—The ball-shaped active electrode (natural size).

tact. The sclera is also seen to be flattened out in a peculiar manner near the treated area. The heating being completed in the manner described, trephining of the sclera is carried out within the treated area with an Elliot trephine. This is preferably done at a place corresponding to the most dependent area of the detachment. On carefully dividing the exposed choroid the subretinal contents escape, whether these are made up of a more or less thin fluid or more normal vitreous humor. No bleeding occurs at the trephining wound, probably owing to coagulation of the blood as a result of the preliminary endothermal treatment. The escaping fluid or vitreous humor is washed away with physiologic solution of sodium chloride. Trephining is carried out partly with the view of removing subretinal fluid, and partly to prevent a subsequent increase of intra-ocular tension or reaccumulation of subretinal fluid. When the operation has been completed in the manner described, the resected muscles and conjunctiva are sutured.

The patient is kept in bed with both eyes bandaged for a fortnight. The head is placed as far as possible in such a position that the detached area lies inferiorly, with the view of furthering important mechanical factors that may help to approximate the retina to the eyeball. In the case of a favorable result the patient is then kept in bed for another

eight to fourteen days, but without bandage. Atropine is given as long as the eye is inflamed.

For a few hours after the operation the patient is often troubled with fairly severe discomfort, with nausea, aching pain in the eye and vomiting. I think that I have been able to reduce this discomfort by giving narcotics (morphine), together with a sea-sickness remedy containing atropine just before, and an hour after, the operation. As a rule the pain soon ceases.

The eyes bear the operation exceedingly well. For a few days after the operation chemosis is often present; however, it soon disappears, and a fortnight after the operation the eyes are, as a rule, free from inflammatory reaction. However, some injection of the conjunctiva remains for rather a long time.

At first the operation was carried out over a very large area of the sclera, corresponding approximately to the extent of the retinal detachment. Nowadays in cases of complete or extensive detachment I confine the operation to that quadrant or portion of the globe within which the hole is located. *No precise localization of the site of the hole is necessary.* In cases in which no hole can be demonstrated, and in cases of large detachments, the electro-endothermal treatment is carried out over a larger area, but particularly within that part where the detachment first came into evidence. To this end the history may be of great value.

The endothermy apparatus that I have made use of is Siemen's "Thermoflux K."

In the Eye Clinic of the Serafimer Hospital I have performed the operation on forty patients since 1930, through the courtesy of the former head of the clinic, Prof. A. Dalen, and its present chief, Prof. J. W. Nordensen. During the same period fifty-five patients with retinal detachment were admitted to the clinic. Those not operated on were discharged because of the hopelessness of their condition, the detachment being of too long standing or complications of one or another kind having arisen. At first, however, I had no intention of selecting any particular type of case; therefore, nearly every patient was operated on with a view to ascertaining what the operation might accomplish, though I was conscious of the hopeless outlook for some.

Efforts to distinguish operatively favorable cases from unfavorable ones merit every consideration, and perhaps a greater experience in operative practice will gradually help me to arrive at an opinion on this point. For the present, however, I am fairly at a loss in this respect. All I know is that the chance of success with operation becomes smaller the longer the detachment has lasted. Those cases seem to offer the best prospect in which the retinal detachment has not been present for more than a few weeks. The chance of operative success seems to be

less favorable for elderly people (over 50 or 60 years), even if the actual age cannot be of any decisive importance.

No serious complications have, as a rule, occurred. The eyes generally stand the operation extremely well. Bleeding occurs relatively rarely. Opacities of the vitreous arise in some cases, but they generally clear up after a longer or shorter space of time.

In one patient—the only one on whom I have operated elsewhere and with another endothermy apparatus—complications ensued, probably some infection or thrombosis with features simulating tenonitis, hemorrhage in the vitreous, amaurosis and, later, beginning atrophy of the globe. Fortunately, this case was the only one of its kind.

Changes similar to those of choroiditic foci in the macular region, observed by J. Meller [3] after operations of this nature, I have seen only once (case 36). Whether the operation was to blame for this result or whether it was due to the nearly completed retinal detachment, in this case overhanging the macula, cannot, I suppose, be ascertained. In a few other cases (cases 5, 33 and 40) macular changes were present prior to the operation.

In one case (case 18), in which the operation resulted in complete reattachment of the wholly detached retina, the optic disk, not previously discernible, proved to be pale and markedly excavated in a glaucomatous manner; vision was much reduced, the visual field restricted and the tension slightly raised. It is inconceivable that these papillary changes, observed a fortnight after the operation, when the eye was free from any inflammatory reaction, could have been caused by the operation. Glaucomatous changes have not been observed in any other patients operated on.

Before operation in one case (case 20) there were, besides detachment of the retina, signs of iridocyclitis, with injection and cloudy aqueous. The operation was successful; the retina became reattached and the eye paler. After some time there were again signs of iridocyclitis of a chronic type, but no retinal detachment. The vitreous became cloudy and the eye was enucleated on account of pain. Prolonged inflammatory conditions have not been observed in any other cases.

Immediately after the operation the retina is still seen to be detached, often to a greater extent than before the operation; but generally from five to eight days after the operation in favorable cases the retina begins to resume its proper place. In successful cases large atrophic areas with pigmented spots can be seen in the fundus corresponding to the treated area. Such changes are sometimes seen within a confluent area, at other times only within small separate patches. These changes, reminding one of old choroidal foci, often appear at first to be prominent, particularly in the periphery, but the retina is not detached.

3. Meller, J.: Ztschr. f. Augenh. **75**:207, 1931.

TABLE 2.—*Data on Patients Operated on by*

Case; Patient	Age; Sex	Extracts from History	Date of Admission	Condition on Admission	Date of Operation
1 A. D.	20 M	Moderate myopia; symptoms of retinal detachment in left eye for 1 month	12/14/29	O.S.V. = 0.2 (—4.00) O.D.V. < or = 0.6 (—6.00 ?) O.S.: Large detachment temporally; no visible hole O.D.: Normal	1/25/30
2 E. H.	40 M	Moderate myopia; symptoms of retinal detachment in right eye for 2 months. 2/17/30: Gonin's operation performed, with negative result	2/ 6/30	O.S.V. = 1 (—5.50) O.D.V. = 0.2 (—5.50) O.D.: Large detachment in whole lower half of fundus; peripherotemporal hole	3/ 3/30
3 H. H.	30 F	No myopia; symptoms of detachment in right eye for 6 months	3/ 3/30	O.S.V. = 0.9 (+1.00) O.D.V. = 0.1 (+4.00 ?) O.S.: Small circumscribed peripherotemporal detachment O.D.: Large detachment in whole lower temporal quadrant; below and temporally, a large hole	3/27/30
4 K. H.	67 F	No myopia; markedly impaired vision for 12 months, especially in left eye	4/ 9/30	O.S.V. < or = 1/60; no improvement by correction O.D.V. < or = 0.4 (+2 ⊂ +2.75°) O.S.: Large detachment in upper temporal quadrant; hole ? O.D.: Smaller detachment; hole	4/10/30
5 B. B.	16 M	No myopia; symptoms of detachment in right eye for 2 months	4/24/30	O.S.V. = 1 (+0.50) O.D.V. = 0.1 (+0.50) O.D.: Large detachment in temporal upper quadrant, involving macula; peripheral hole O.S.: Normal	5/ 3/30
6 M. L.	67 F	No myopia; impaired vision in both eyes for last 2 years; during last few weeks, right eye rapidly became worse	4/ 7/30	O.S.V. < or = 0.3 (—0.50) O.D.V. = 2/60; no improvement by correction O.S.: Glaucomatous disk; no rise of tension O.D.: Poor red reflex; ablatio below	5/ 3/30
7 E. P.	38 M	Marked myopia; vision in right eye getting rapidly worse during last week	6/ 5/30	O.S.V. = 0.1 (—14.00 ⊂ —2.00, 45°) O.D.V. = 1/60; no improvement by correction O.D.: Large detachment above, reaching over macula; peripherally, multiple ruptures O.S.: Normal	6/ 6/30
8 J. S.	78 M	No myopia; patient discovered accidentally a fortnight before that vision was much reduced in the right eye	8/ 8/30	O.S.V. = 0.3 (+3.00) O.D.V. = counting fingers, close to eye downward and temporally O.D.: Complete ablatio; hole not observed O.S.: Numerous opacities in vitreous	8/ 8/30
9 V. M.	42 F	Moderate myopia; symptoms of detachment in left eye for 6 weeks. Treatment: rest in bed	8/10/30	O.S.V. < or = 0.2; no improvement by correction O.D.V. = 0.7 (—7.00 ⊂ —0.50, 90°) O.S.: Large detachment in lower half; no hole O.D.: Normal	8/12/30
10 A. S.	52 M	No myopia; always saw worse with right eye; symptoms of detachment in right eye for 3 weeks	8/23/30	O.S.V. = 1 (—0.50, 90°) O.D.V. = hand movements O.D.: Complete detachment; large hole extending nasally upward O.S.: Normal	8/29/30
11 A. E.	47 F	Well marked myopia; symptoms of detachment in left eye for about 5 days	9/ 5/30	O.S.V. = hand movements in small excentric temporal field O.D.V. = 0.4 (—10.00 ⊂ —1, 180°) O.S.: Complete detachment with large peripherotemporal hole	9/13/30
12 F. L.	53 M	No myopia; trauma to right eye, 9 days before	9/12/30	O.S.V. = 1 (+1.00) O.D.V. = hand movements O.D.: Complete ablatio; hole extending upward and nasally	9/13/30
13 E. M.	45 F	Gonin's operation in September, 1929; no result; asked for another operation	9/13/30	O.S.V. = 0.2 (—22.00) O.D.V. = hand movements O.D.: Complete ablatio; hole in upper nasal periphery	9/15/30

Condition After Leaving Bed	Follow-Up Examination	Condition on Follow-Up Examination	Period of Observation	Result	Comment
2/13/30: O.S.V. = 0.2 (−4.00); no detachment	6/ 9/31	O.S.V. = 0.5 (−4.50 ⌒ −1.00, 180°); read Jaeger type 1 (−1.00); no detachment visible	1 yr., 4½ mos.	Positive	
2/27/30: O.D.V. < or = 0.3 (−8.00); no detachment visible	6/11/31	O.D.V. < or = 0.3 (−8.00); read Jaeger type 1 (−5.00); no detachment visible	1 yr., 3½ mos.	Positive	
6/2/30: O.D.V. = 0.1 (−1.00); hemorrhage in vitreous; no ablatio visible	6/ 2/31	O.D.V. = 0.2 (−0.50); no ablatio visible	1 yr., 2 mos.	Positive	
5/6/30: O.S.V. = 0.1 (+7.00?) detachment still persists at the site of operation	.....	According to written statement in May, 1931, patient in worse condition since coming home	.....	Negative	
6/6/30: O.D.V. < or = 0.3 (±0); no detachment visible	6/ 8/31	O.D.V. < or = 0.3 (±0); read Jaeger type 7 without correction; no detachment; in macula, "choroiditic" changes	1 yr., 1 mo.	Positive	
6/1/30: Condition unchanged	10/15/30	O.D.V. = hand movements; large detachments; in parts, however, retina was in its place	5 mos.	Negative	
6/20/30: O.D.V. = 0.1 (−16.00); no detachment visible	6/ 2/31	O.D.V. < or = 0.3 (−16.00 ⌒ −2.00, 35°); read Jaeger type 1 (−10.00); no detachment visible	1 yr.	Positive	
Hemorrhage in vitreous	6/10/30	V. = perception of light; complete ablatio	2 mos.	Negative	
Large hemorrhage pervading vitreous	2/23/31	O.D.V. = hand movements; complete ablatio	6 mos.	Negative	
9/12/30: V. < or = 0.1 (−3.00 ⌒ −3.00, 180°); no ablatio visible	Not reexamined	According to subsequent statement, well marked aggravation about 1 mo. after discharge	.....	Negative (recurrence ?)	Recurrence or relapse
9/29/30: O.S.V. = 0.1 (−9.00 ⌒ −2.00, 80°); no ablatio visible	11/20/30	O.S.V. = 1-2/60; retina detached here and there; much haziness in vitreous	2 mos.	Negative (recurrence)	Recurrence or relapse
Condition unchanged	11/12/30	Condition unchanged; complete ablatio	2 mos.	Negative	
9/30/30: Complete ablatio	10/ 6/30	O.D.V. = 2/60; apparently complete ablatio	5 wks.	Negative	

TABLE 2.—*Data on Patients Operated on by Electro-Endothermy*

Case; Patient	Age; Sex	Extracts from History	Date of Admission	Condition on Admission	Date of Operation
14 G. W.	35 M	Complicated case showing adherent leukoma; symptoms of detachment in left eye for 6 months	9/17/30	O.S.V. = 1/60 O.D.V. = amaurosis O.S.: Large detachment; poor view of fundus	9/19/30
15 F. A.	60 F	Well marked myopia; symptoms of detachment in left eye for 3½ months; Gonin's operation performed previously (Lindberg)	10/ 1/30	O.S.V. = hand movements in a small temporal field O.D.V. = 0.4 (−10.00 ⊃ −1.00, 75°) O.S.: Complete ablatio; small hole (?) extending upward nasally, in the far periphery	10/ 2/30
16 M. A.	46 F	No myopia; symptoms of detachment in right eye for 3½ months	10/10/30	O.S.V. = 1 (+0.50) O.D.V. = 0.5 (+0.75) O.D.: Large ablatio in lower half; large peripheral hole	10/11/30
17 K. E.	33 M	No myopia; symptoms of detachment in left eye for 6 weeks; no symptoms in right eye	10/20/30	O.S.V. = 0.6 (±0) O.D.V. = 1 (+0.50) O.S.: High ablatio in upper temporal quadrant; long hole peripherally O.D.: Same condition as in left eye, but less well marked	10/11/30
18 F. K.	50 M	No myopia; patient thought he had seen well before; about 14 days before examination, he noticed vision impaired in left eye	10/ 1/30	O.S.V. = hand movements O.D.V. = 1 (−0.50, 75°) O.S.: Complete ablatio covering disk; no hole visible O.D.: Normal	10/20/30
19 I. I.	40 M	Moderate myopia; right eye always worst; symptoms of detachment in right eye for 1½ weeks	11/ 2/30	O.S.V. < or = 1 (−4.00 ⊃ −2.00, 90°) O.D.V. = hand movements O.D.: Large detachment in the whole upper half; large hole upward and temporally	11/ 3/30 11/28/30
20 H. N.	53 M	No myopia; for past 3 weeks, injection and impaired vision in the right eye	12/29/30	O.S.V. = 0.9 (+0.50) O.D.V. = 0.7 (−1.00) O.S.: Normal O.D.: Pericorneal injection; aqueous not clear (slit-lamp); upward, high ablatio in folds; no hole	1/ 5/31
21 K. G.	15 M	No myopia; no trauma; about 2 months before, patient noticed impaired vision in left eye. Scleral puncture performed (Forsmirk)	1/ 8/31	O.S.V. = perception of light O.D.V. = 1 (+1.00) O.S.: Aqueous not clear (Gullstrand's slit-lamp); complete ablatio; no hole visible; numerous opacities in vitreous	1/16/31
22 O. M.	51 F	Moderate myopia; symptoms of detachment in right eye for about 3 months	1/17/31	O.S.V. = 0.9 (−5.00) O.D.V. < or = 0.3 (−5.00) O.D.: Large detachment nasally and upward; small peripheral hole	1/19/31
23 H. B.	60 M	No myopia; symptoms of detachment in left eye for 14 days	1/19/31	O.S.V. = 2/60 O.D.V. = 1 (+1.50) O.S.: Large detachment in whole temporal half; no hole visible	1/22/31 and 2/19/31
24 G. L.	50 M	Moderate myopia; 10 years before, ablatio in right eye with rapid loss of sight; symptoms of detachment in left eye for about 3 months	1/24/31	O.S.V. < or = 1 (−4.00 ⊃ −0.75, 180°) O.D.V. = amaurosis O.S.: Straight upward, high ablatio; peripheral hole O.D.: Cataract; status glaucomatosus	1/26/31
25 G. L.	50 M	Same patient as in case 24; recurrence for past 8 days	7/13/31	O.S.V. < or = 1 (−3.50 ⊃ −0.75, 180°) O.D.V. = amaurosis O.S.: Large ablatio downward, without visible hole above, retina is well in place	7/15/31
26 F. N.	28 F	No myopia; symptoms of detachment in right eye for more than 3 months	3/ 4/31	O.S.V. = 1 (+1.25) O.D.V. =1/60 in a small low field O.D.: Large detachment in whole lower half; below, large peripheral hole	3/24/31
27 G. L.	15 F	Marked myopia; symptoms of detachment in left eye for 14 days	3/13/31	O.S.V. = 5/36 (−10.00 ⊃ −3.00, 5°) O.D.V. = 0.1 (−10.00) O.S.: Myopic, macular changes and peripheral pigmented areas O.D.: Large detachment temporally; no visible hole	3/24/31

Condition After Leaving Bed	Follow-Up Examination	Condition on Follow-Up Examination	Period of Observation	Result	Comment
12/20/30: Complete ablatio; V. = perception of light	1931	Condition unchanged	.....	Negative	
10/19/30: Condition unchanged	.....	.....	1 mo.	Negative	
11/5/30: V. = 0.4 (+0,50); no ablatio visible; slight amount of blood in vitreous	6/13/31	O.D.V. < or = 1 (—0.50); read Jaeger type 1 +1.50; no ablatio visible	8 mos.	Positive	
A subretinal hemorrhage extending down over macula; V. = 0.1	12/16/31	O.S.V. < or = 0.4 (+1.00 ⌒ —2.00, 180°); O.D.V. = 1 (+0.50) O.S.:Low ablatio; definite delimitation impossible O.D.: Same as on discharge	14 mos.	Positive and negative	Clearly marked tendency toward spontaneous healing
11/13/30: V. = 0.5/60; no ablatio; pale glaucomatous disk	6/ 7/31	O.S.V. = hand movements, temporally; O.D.V. = 1 (—0.50, 75°) O.S.: No ablatio; status glaucomatous	7 mos.	Positive	Anatomically good result
12/20/30: No ablatio visible	12/26/30	O.D.V. = 0.5/60; large detachment temporally	.....	Negative	
1/23/31: O.D.V. < or = 0.7 (—2.00); still some injection of globe	6/ 6/31	O.D.V. = 0.2 (—1.00); increased injection; iridocyclitis; no ablatio visible	6 mos.	Positive	Subsequent enucleation on account of iritis and pains
Condition unchanged	3/ 2/31	Condition unchanged; V. = perception of light	6 wks.	Negative	
2/18/31: O.D.V. < or = 0.4 (—6.00); no detachment visible	12/ 9/31	O.D.V. < or = 0.7 (—5.50); read Jaeger type 1 without correction; no detachment visible	10½ mos.	Positive	
3/2/31: O.S.V. = 2/60; complete ablatio	9/ 9/31	Complete ablatio	7½ mos.	Negative	
3/2/31: O.S.V. < or = 0.6 (—4.00); some blood in vitreous; no ablatio visible	5/28/31	O.S.V. < or = 1 (—3.50 ⌒ —0.75, 180°); no ablatio visible	4 mos.	Positive	Patient well until about 7/5/31, when signs of recurrence arose; see case 25
Condition unchanged after three operations; then progressive improvement	.....	.....	.....	Negative	Recurrence with ablatio in opposite part of retina
4/22/31: O.D.V. = 0.2 (—2.00, 150°); no ablatio visible	12/10/31	O.D.V. < or = 0.5 (+0.50 ⌒ —2.00, 135°); read Jaeger type 3 with correction; no detachment visible	8½ mos.	Positive	
4/9/31: V. = hand movements; complete ablatio	.....	.....	.....	Negative	

TABLE 2.—*Data on Patients Operated on by Electroendothermy*

Case; Patient	Age; Sex	Extracts from History	Date of Admission	Condition on Admission	Date of Operation
28 A. B.	31 F	No myopia, but vision always worse in right eye; symptoms of detachment in right eye for 1 week	4/ 2/31	O.S.V. = 0.9 (+0.50 ⊂ −1.00, 15°) O.D.V. = hand movements O.D.: Complete ablatio upward; large peripheral hole upward and temporally	4/ 2/31
29 M. C.	68 M	No myopia; symptoms of detachment in right eye for 5 days	4/15/31	O.S.V. = 0.7 (+1.00) O.D.V.: = 1/60 O.D.: Large ablatio in whole upper half; upward and temporally a hole the size of disk	4/18/31
30 L. E.	27 M	No myopia; trauma to left eye 1 month before	4/22/31	O.S.V. = 0.3; no improvement by correction O.D.V. < or = 1 (±0) O.S.: Ablatio in lower temporal quadrant; no hole visible	4/25/31
31 E. P.	33 F	No myopia; symptoms of detachment in left eye for 14 days	5/ 6/31	O.S.V. = 0.3 (+1.00) O.D.V.: = 1 (+0.50, 180°) O.S.: Ablatio in lower temporal quadrant; large hole peripherally and downward temporally	5/ 8/31
32 M. S.	22 F	Slight myopia; symptoms of detachment in left eye for 6 days	5/29/31	O.S.V. = 0.2-0.3 (−2.00) O.D.V. < or = 0.7 (−2.75 ⊂ −2.50, 30°) O.S.: Large detachment in whole upper half of fundus; large hole peripherally	6/ 2/31 and 7/ 7/31
33 E. E.	44 F	Slight myopia; impaired sight of right eye accidentally (disk) 2 months before	6/15/31	O.S.V. < or = 1 (−0.75) O.D.V. < or = 0.1 (−3.00 ?) O.S.: Normal O.D.:Low detachment in whole lower half of fundus; no hole visible; streaks of pigment also in macula	6/16/31
34 F. A.	43 M	Slight myopia; symptoms of detachment in left eye for 14 days	6/22/31	O.S.V. = counting fingers close to eye O.D.V. = 0.6 (−0.75, 20°) O.S.: Large fluttering detachment upward, overhanging disk and macula O.D.: Opacities in lens and vitreous	6/23/31
35 S. P.	44 F	No myopia; symptoms of detachment in left eye for 10 days	7/ 8/31	O.S.V. = 1/60 O.D.V. < or = 1 (+0.50) O.S.: High fluttering ablatio in temporal upper quadrant covering macula; small peripheral hole O.D.: Normal	7/10/31
36 G. J.	40 M	No myopia; symptoms of detachment in left eye for 14 days	7/27/31	O.S.V. = 0.50/60 O.D.V. = 1 (+1.00) O.S.: Almost complete, high fluttering detachment; hole the size of disk, temporally O.D.: Normal	7/29/31
37 E. Z.	47 F	Moderate myopia; July, 1929, ablatio retinae in left eye and rapid loss of sight; symptoms of detachment in right eye for 4 days	8/ 6/31	O.S.V. = amaurosis O.D.V. = 2/60 (−7.00) O.S.: Complete old ablatio O.D.: Large high ablatio upward, covering macula and disk; peripheral hole above	8/ 7/31
38 M. L.	60 M	No myopia; symptoms of detachment in right eye for 14 days	8/20/31	O.S.V. = 1 (+1.00, 90°) O.D.V. = hand movements O.S.: Normal O.D.: Large detachment upward, with large peripheral hole	8/21/31
39 A. E.	60 F	Marked myopia; symptoms of detachment for at least 5 months	9/ 9/31	O.S.V. = counting fingers close to eye O.D.V. = 0.2 (−16.00) O.S.: Large ablatio downward; no hole visible O.D.: Myopic changes in fundus	9/11/31
40 L. A.	22 F	Slight myopia; symptoms of detachment in left eye for 6 months	9/14/31	O.S.V. < or = 0.1 (−2.00 ⊂ −3.50, 180°) O.D.V. < or = 1 (−2.75 ⊂ −1.50, 25°) O.S.: Low ablatio in whole lower half; in macular region, "choroiditic" changes; small peripheral hole below O.D.: Normal	9/14/31

for Detachment of the Retina—Continued

Condition After Leaving Bed	Follow-Up Examination	Condition on Follow-Up Examination	Period of Observation	Result	Comment
4/25/31: O.D.V. = 0.1 (−4.00 ⌒ −2.00, 30°); no ablatio visible	5/ 7/31	Nearly complete ablatio	.....	Negative	Relapse
5/2/31: V. < or = 1/60; nearly complete ablatio	.....	.....	.....	Negative	
6/2/31: V. < or = 0.5; no improvement by correction; blood in vitreous; no definite ablatio	11/23/31	O.S.V. < or = 0.6 (±0); read Jaeger type 1 without correction; no ablatio, but extensive atrophic areas reaching fairly far to the centrum	7 mos.	Positive	Posttraumatic ablatio
6/8/31: O.S.V. < or = 0.5 (+1.50); no ablatio visible	11/17/31	O.S.V. < or = 1 (+1.00, 135°); read Jaeger type 1; no ablatio visible	>6 mos.	Positive	
Relapse one week after first operation; another operation performed. 5/8/31: O.S.V. = 0.2 (−3.00 ⌒ −0.75, 180°); no ablatio	11/23/31	O.S.V. < or = 0.4 (−2.75 ⌒ −2.50, 35°); read Jaeger type 1 (−2.50, 35°); no ablatio visible	5½ mos.	Positive	Relapse after first operation
7/20/31: V. = 0.1 (−3.00); no detachment	11/20/31	O.D.V. = 0.3 (−1.50 ⌒ −2.00, 40°); read Jaeger type 6 (−2.00, 40°); in macula, streaks of pigment; no detachment	5 mos.	Positive	
7/25/31: O.S.V. = 0.1 (−1.00); no detachment	11/20/31	O.S.V. < or = 0.4 (−1.00); read Jaeger type 4 without correction; no ablatio visible	5 mos.	Positive	
8/15/31: O.S.V. = 0.4 (+0.75 ⌣ +1.25, 90°); no detachment	11/23/31	O.S.V. = 0.4 (+0.50, 90°); read Jaeger type 2 (+1.50 ⌒ +0.50, 90°); no detachment visible	4½ mos.	Positive	
8/28/31: O.S.V. < or = 0.5 (−0.50); no detachment	11/25/31	O.S.V. = 0.1 (−1.00); no detachment; centrally, a "choroiditic," somewhat prominent, diffuse area, the size of disk	4 mos.	Positive	A central "choroiditic" or similar process has impaired vision
9/9/31: O.D.V. < or= 0.6 (−6.00 ⌒ −1.50, 15°); no visible detachment	12/ 4/31	O.D.V. < or = 1 (−6.00 ⌒ −1.50, 15°); read Jaeger type 1 (−4.00 ⌒ cyl.); no ablatio visible	4 mos.	Positive	
9/23/31: Condition unchanged	.....	.....	.....	Negative	
9/26/31: Condition unchanged	.....	.....	.....	Negative	
10/30/31: O.S.V. = 0.2 (−3.00 ⌣ −3.50, 180°); no detachment visible	12/22/31	O.S.V. < or = 0.3 (−3.50 ⌒ −3.50, 180°); read Jaeger type 3 with cylindric lenses; no detachment visible; small atrophic spots in macular region	3 mos., 7 days	Positive	

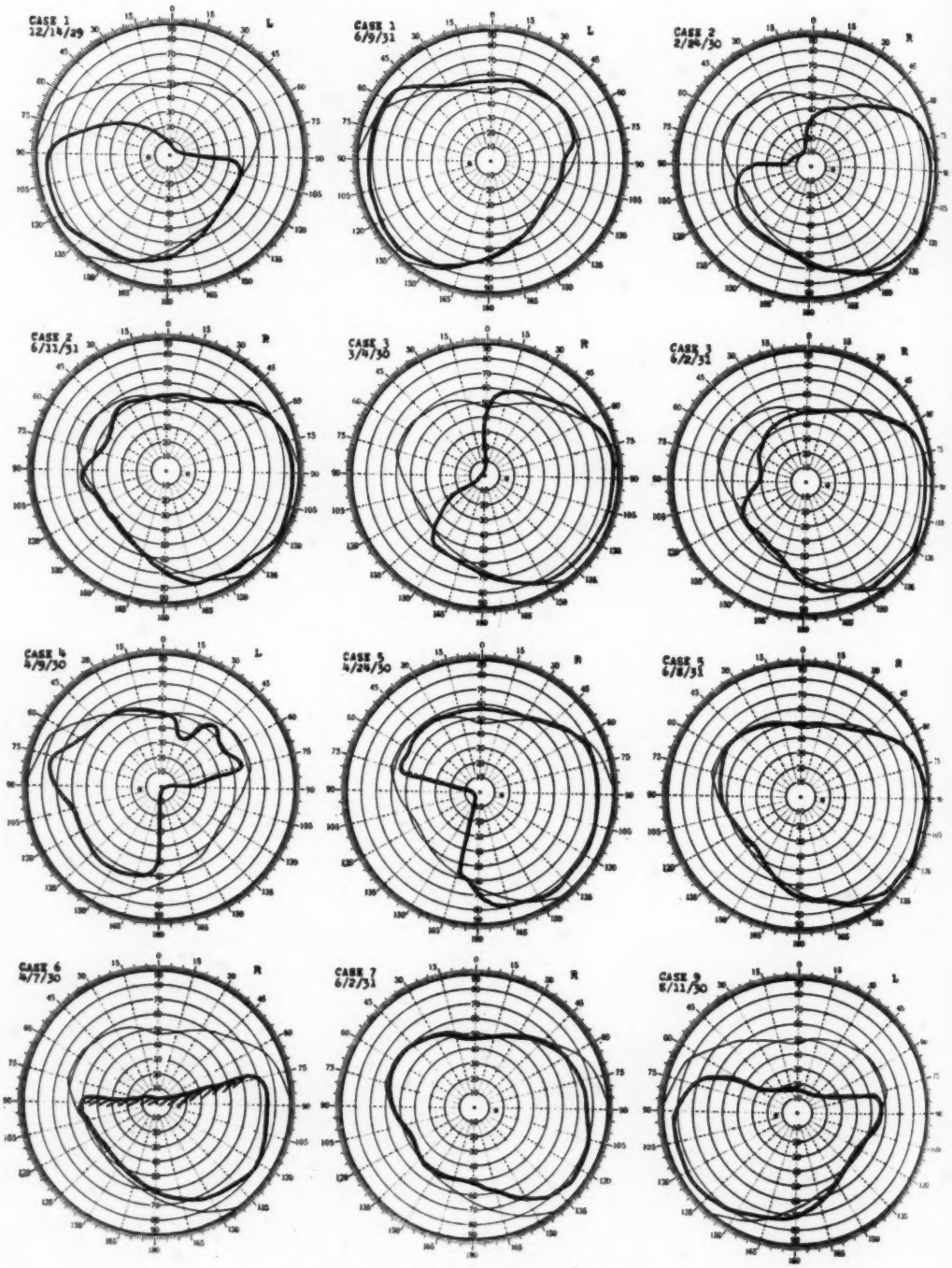

Fig. 2.—Visual fields in cases 1 to 9.

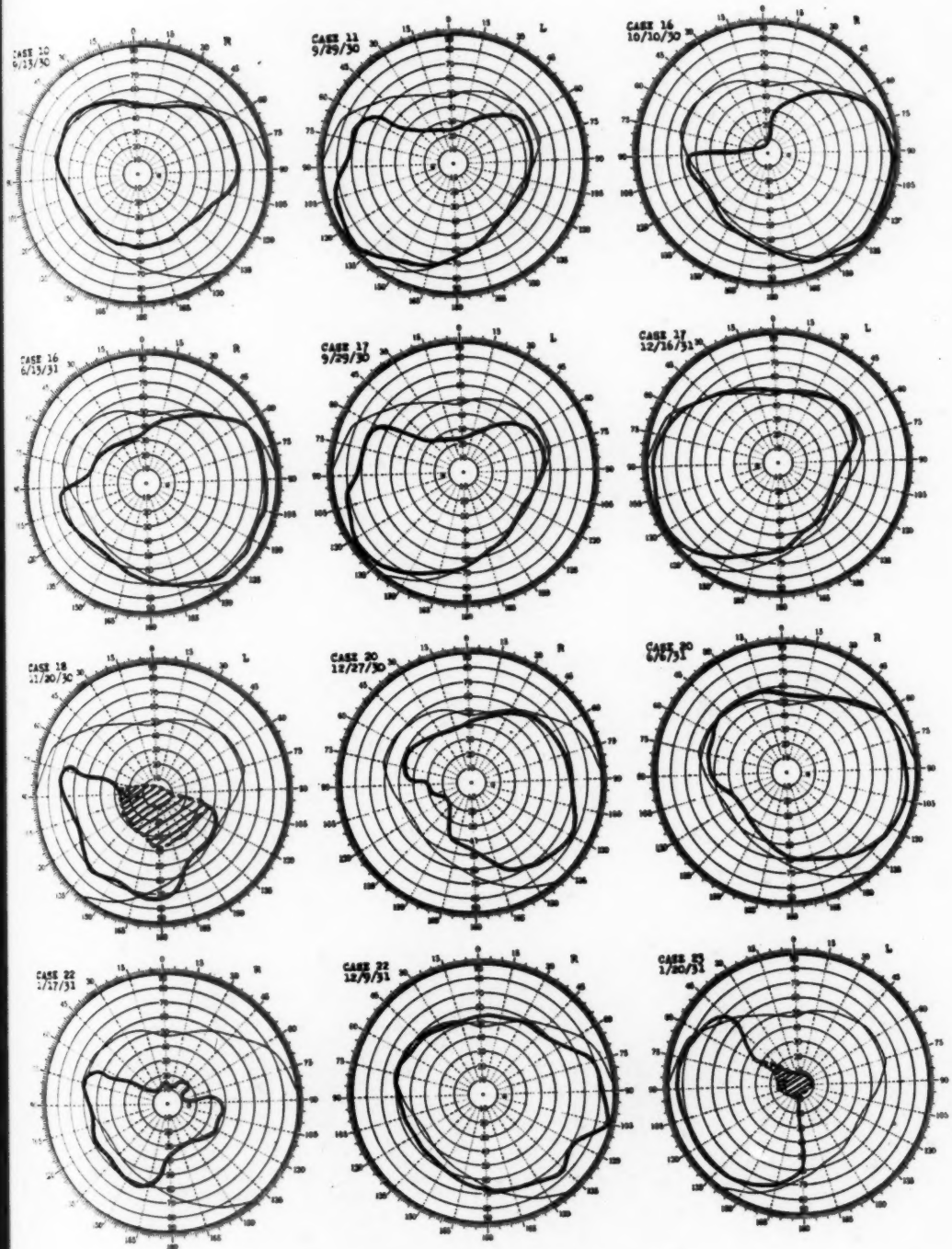

Fig. 3.—Visual fields in cases 10 to 23.

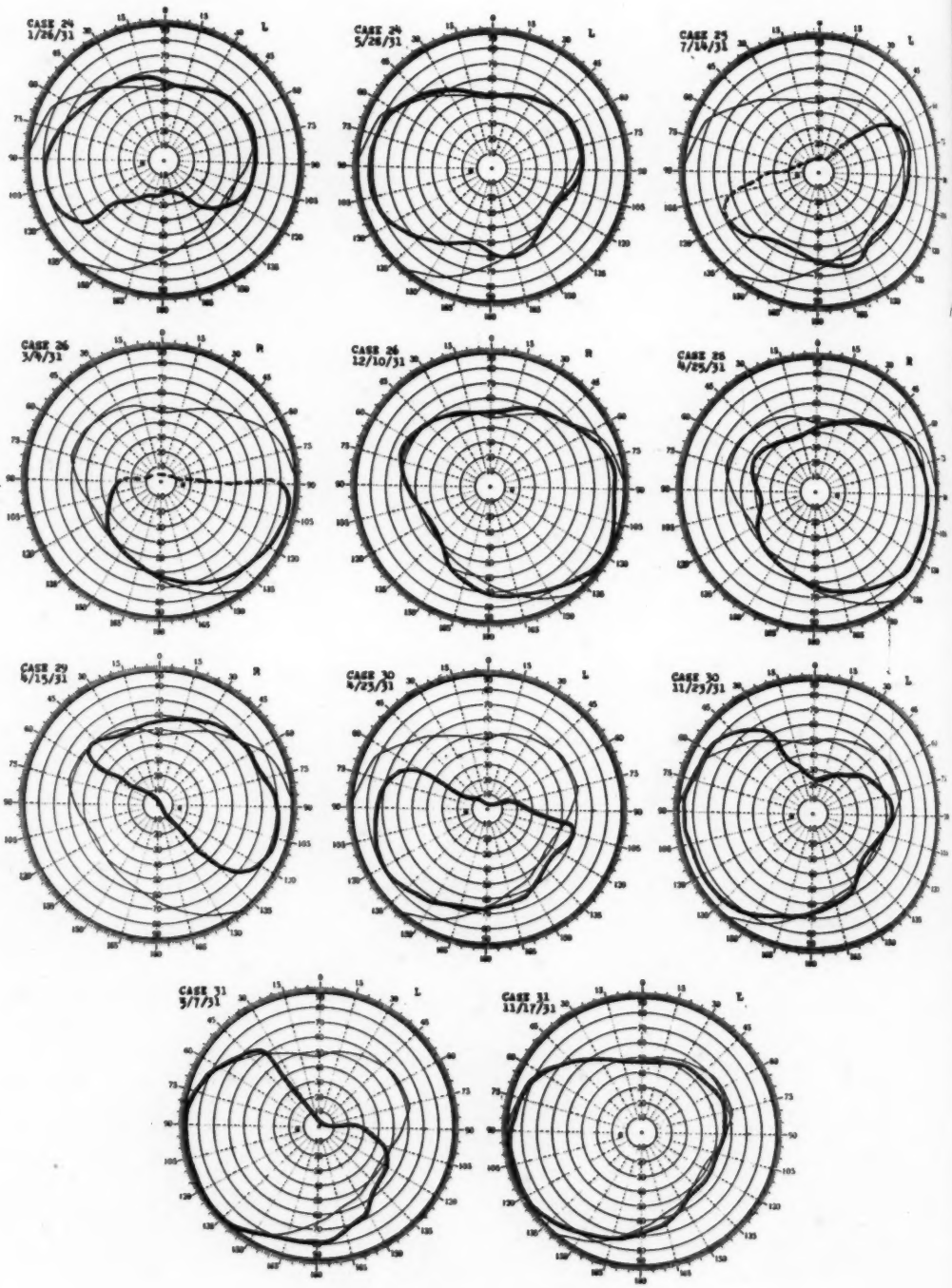

Fig. 4.—Visual fields in cases 24 to 31.

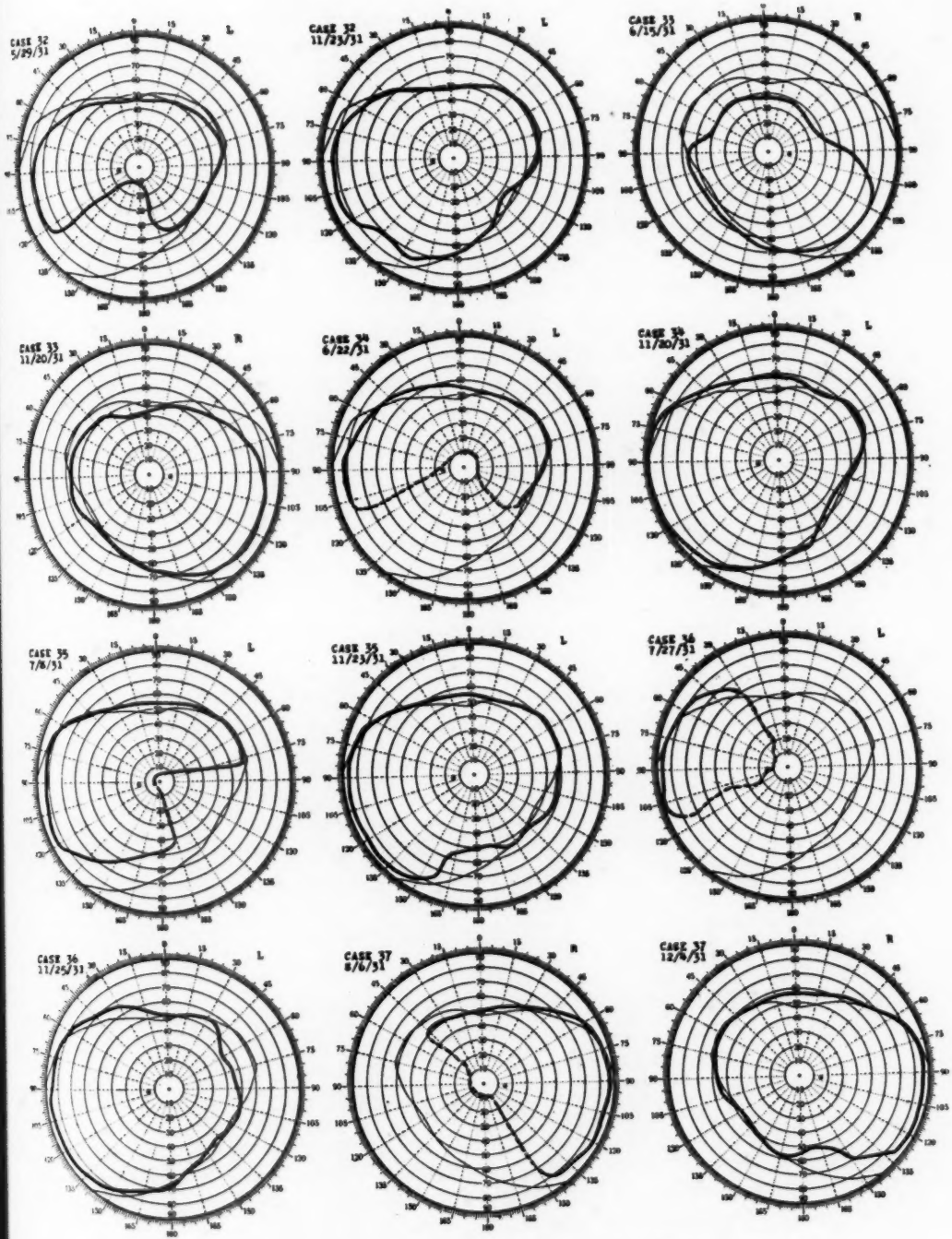

Fig. 5.—Visual fields in cases 32 to 37.

In cases in which the operation has been successful, vision improves as a rule materially, though in varying degrees, probably depending on the extent of damage sustained by the retina as a result of the detachment. The field of vision always improves in successful cases, and, extraordinarily enough, the limits of the field of vision are only moderately or slightly, and in some cases not at all, contracted as a result of the postoperative "choroiditic" changes in the periphery of the fundus. (The fields of vision, however, have been tested only with a large white 10 to 15 mm. object and in a good light with an ordinary McHardy perimeter.)

The forty cases in which I performed an operation at the Eye Clinic of the Serafimer Hospital are presented in table 2. In all successful cases a follow-up examination was performed. All successful cases have been denoted "positive." By this is meant complete replacement of the retina without any ablatio being discernible anywhere in the fundus with the ophthalmoscope. Cases showing slight improvement or partial reattachment of the retina have been referred to as "negative." In one case (case 24) there was a recurrence five months after the operation, with detachment of the retina at quite a different place from that of the first detachment. Renewed operation was not successful. It has seemed to me most proper to reckon this as two cases, one positive and one negative. Particulars and results in the individual cases are given in table 2.

In this series of forty cases there were twenty (50 per cent) successful or positive cases, and this in spite of its being encumbered with a great many hopeless cases that, in the light of later experience, should never have been operated on.

The advanced age of the patient, long-standing nature of the detachment, operations performed elsewhere and complications of one kind or another, contraindicated surgical intervention in the following cases:

Case 4: A patient, aged 67, with symptoms present probably for a year.

Case 6: A patient, aged 67, with symptoms that had lasted for an indefinite time.

Case 8: A patient, aged 78, with symptoms that had probably lasted for a long time.

Case 13: An operation performed elsewhere twelve months earlier by Gonin's method without any results.

Case 14: A complicated case showing adherent leukoma in both eyes, amaurosis of one eye, probably a gonorrheal infection, and symptoms of retinal detachment in the other eye for at least six month. An operation was performed at the patient's own urgent request.

Case 15: Operation performed elsewhere by Gonin's method without result.

Case 21: A case showing in places the appearance of retinitis proliferans. The patient had undergone scleral puncture elsewhere. The detachment was complete and no hole could be observed.

Case 28: A case in a man, aged 68, which, except for the advanced age of the patient, did not seem unfavorable.

Case 38: A patient, aged 60, with symptoms of retinal detachment for the past five months.

With the possible exception of the two last-mentioned cases these patients should be excluded from the list in estimating the value of the operation. There then remain thirty-three cases that, according to my present experience, might be said to offer from the outset a chance of successful operative treatment. Calculated in this way, a better result, of course, is obtained: Of thirty-three cases twenty have shown successful results (i. e., about 60 per cent).

The number of cases is too small and the time of observation of the patients last operated on is too short to permit of any final judgment as to the value of the operative method described here. This much, however, may be said: The results are encouraging. The method is new, and still further improvement in the technic may be hoped for. A more reliable dosage of heat would be particularly desirable. It is probably of some importance that the appropriate amount of heat be used. Yet the dosage becomes a matter of experience in this operation as well as in Gonin's. No decisive importance can be ascribed to the amperemeter reading, as important factors, such as tissue resistance, moisture, contact, etc., vary from case to case.

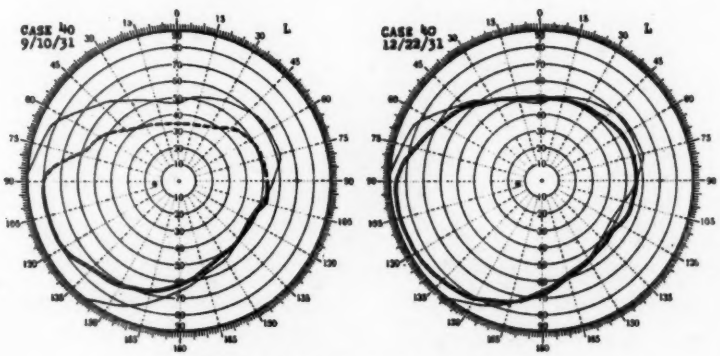

Fig. 6.—Visual fields in case 40.

There would seem to be no doubt that this operation has a beneficial effect on the retinal detachment through the adherence that it brings about between the detached retina and the underlying tissues, a process in which is also included the adherence of the margins of the hole to the underlying tissue. It may be that extensive subsequent atrophy of the choroid may act in a beneficial manner by reason of the fact that the conditions for the formation of fluid from the choroid no longer exist.

Provided that the endothermal treatment yields equally good results in the future, it seems to me that in comparison with Gonin's operation it possesses certain advantages.

By creating a more widespread adherence between the retina and the choroid, fresh detachments, like fresh holes, arise less readily in the area adjacent to the primary lesion. On this account, recurrences arise perhaps less readily and less often, and the operation need not be repeated time after time as is so often the case with Gonin's operation.

Bleeding rarely occurs, probably owing to the thermal effect on the vessels, such as coagulation and thrombosis.

While in Gonin's operation the searing instrument is introduced into the vitreous humor, the thermal effect in electro-endothermy takes place from without, and no cicatricial plug is obtained in and toward the vitreous from which traction can take place on the retina, leading to fresh detachments and holes. These and other views as to the advantages of electro-endothermy have been put forth by J. Meller,[3] who has tried the method successfully in a few cases.

Electro-endothermy is an easy operation to perform and there is no necessity for the precise localization of the hole. The operation is in need of further trial for a clear view of its possible results.

THE FOUNDATION OF THE NEW YORK EYE AND EAR INFIRMARY

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NEW YORK

"To do justice to the merits of those who have gone before, by recording their names, with honorable praise, is a duty we owe them."

The New York Eye and Ear Infirmary began its existence one hundred and eleven years ago, under circumstances that are interesting from different points of view. It seems not inappropriate to recall the general state of affairs of the city and country when, on Aug. 14, 1820, the first patient, one suffering with a fistula of the lacrimal sac, was registered at the Infirmary. The influences of colonial and revolutionary days were as yet strong in the land. There were still living people in New York whose memory carried them back to the Stamp Act Riot of 1765, and many there were who were participants or onlookers in the Doctors' Riot of 1788, which raged for three days. This grew out of indiscretions on the part of medical students and exhumators who preyed on the potters' field and the Negroes' burying ground. Without doubt pensioners of the Revolution who crossed the Delaware with Washington or helped to surround Cornwallis at Yorktown are numbered among the first of the nearly 2,500,000 patients treated at this institution since its foundation. Jefferson and Adams, and other signers of the Declaration of Independence, had a number of years to live, and Napoleon and the Duke of Wellington had not been gathered in, when the Infirmary was born. New York City numbered a little over 150,000 inhabitants. Its northern limits reached not far beyond the City Hall, the rest of the island being open country. No vessel propelled by steam had yet ventured to cross the ocean from this port. A brief seven years before, the

Submitted for publication, Jan. 30, 1932.

An address delivered in abstract before the Section of Ophthalmology of the New York Academy of Medicine, Nov. 17, 1931. Information has been freely drawn from the following sources: Early Minutes and Reports of the New York Eye and Ear Infirmary; An Historical Address at the Commemoration of the 150th Anniversary of the New York Hospital by Edward W. Sheldon; The Development of Ophthalmology in America from 1800 to 1870, by Alvin A. Hubbell (Chicago, American Medical Association Press, 1908); Biographical Sketch of John Kearney Rodgers by Edward Delafield (read before the New York Academy of Medicine, Oct. 6, 1852, and published by its authority); Life of Sir Astley Cooper by Barnsby Blake Cooper; The History and Traditions of Moorfields Eye Hospital by Treacher Collins (London, H. K. Lewis & Co., Ltd., 1929), and The Wills Hospital of Philadelphia by William Campbell Posey and Samuel Horton Brown (Philadelphia, J. B. Lippincott Company, 1931).

harbor was blockaded by a British fleet. Men's conversation was of wars and rumors of war. Slavery existed in New York, and the public whipping post was in use. Many of the houses were of wood, and fires were frequent and disastrous. Illuminating gas had not come into use in New York. There was no system of sewerage, but instead the city's refuse was dumped into the river in the darkness of night. Philadelphia was the medical center of the country and was to continue to be so for another generation and more.

The city of New York boasted of one general hospital, the New York Hospital, founded in 1771, called locally "the Hospital." It was not an era of general hospitals, there being but few in the world. The establishment of a general hospital was an event of such rare occurrence that it was said that prior to the formation of the Charing Cross Hospital in London, in 1822, no new general hospital had been created in that city in eighty years, notwithstanding the fact that the population had doubled. By 1820, Paris had been superseded as the great center of surgery by London. A large body of students from all parts of the world flocked there to witness the practice and surgery of such men as Astley Cooper, Haighton, Cline, Abernethy, Lawrence and Travers. Looking backward to this epoch, it is impossible to separate medicine in America from that in England. America inherited English medicine. In the early decades of the nineteenth century it was still relying on English textbooks and was still attending English universities. It would be particularly difficult to portray the foundation of the New York Eye and Ear Infirmary, which represents the very beginning of scientific ophthalmology in the Western Hemisphere, without considering what was going on in London.

SAILING OF THE FOUNDERS FOR ENGLAND

In 1816, two New Yorkers, Edward Delafield and John Kearney Rodgers, recent graduates of the College of Physicians and Surgeons, set sail for London. They were intent on completing their professional training. They had already enjoyed exceptional opportunities at home, Rodgers having served as surgical resident and Delafield as medical resident at the New York Hospital, during the same period. It happened that while "walking the wards" of the London hospitals, they came on another young American, Edward Reynolds, a graduate of Harvard University. Piecing together the fragments of the past, one learns of still a fourth young American in Europe at the same time, George Frick, of Baltimore, a graduate of the University of Pennsylvania, who had penetrated into the interior as far from the coast as Vienna, where he became a favorite pupil of Beer, the greatest ophthalmologist the world had ever produced. All four of these men were destined to leave their

imprint on American ophthalmology. Among the various hospitals that offered facilities for instruction in London, Delafield, Rodgers and Reynolds were induced to matriculate at the London Infirmary for Curing Diseases of the Eye, which as time wore on came to be known as the Royal London Ophthalmic Hospital (Moorfields). This renowned Charity was founded in 1804 by John Cunningham Saunders, who lived but five years afterward. Its doors, however, were not thrown open to students until 1811. Sir William Lawrence, Mr. Benjamin Travers and Dr. John Richard Farre were the medical officers at this institution, and no less a personage than Sir Astley Cooper was wont to operate on the eye there. It was in an unpremeditated way, while listening to this remarkable coterie at the London Eye Infirmary, that Delafield and Rodgers, first having been made aware of their complete ignorance of the subject, developed their lifelong interest in ophthalmology.

STATE OF OPHTHALMOLOGY IN ENGLAND

Gutherie, the British army surgeon, states contemporaneously, that previous to 1817, no lectures were delivered in Great Britain on diseases of the eye, unless the concise observations that were made in the schools of anatomy on some of the operations are considered as such. The consequence was that a knowledge of these complaints was not presumed to form a part of the education of a surgeon. Without doubt, a similar opinion existed in America. The great number of casualties suffered by the British forces in the Napoleonic wars rendered it imperative for many of the physicians and surgeons of the army and navy to pay greater attention to diseases of the eye. It was related of Saunders that he was moved to found the London Eye Infirmary out of compassion for the dreadful sufferings from ophthalmia of the British troops on their return from the expedition in Egypt. It was in this way, according to Gutherie, that many medical men became convinced of the propriety of their acquiring a knowledge of these diseases, by the same means, and in the same manner, as were found necessary in the study of medicine and surgery in general.

Sir William Travers, speaking also contemporaneously, makes the assertion, that no general surgeon in England prior to himself ever ventured to give more than cursory attention to diseases of the eye, because of the fear of being disqualified, in the opinion of the public, for ordinary practice, should he acquire a reputation for proficiency in the treatment of diseases of the eye. It was common practice for reputable practitioners in England and America, realizing their own ignorance, to refer patients suffering from diseases of the eye to "oculists," who were mere quacks without knowledge of anatomy, pathology or therapeutics. Ophthalmology was in a parlous state in

England and her late dependencies in America. However, the reverse was true in the German-speaking world. Barth founded the Vienna School of Ophthalmology in 1773, spoken of as the greatest event in the history of ophthalmology. Since that time the study of diseases of the eye had received marked attention and had made great progress at the hands of the German medical profession. Beer, who was Barth's successor, wrote a master book on the subject from observations gleaned in wards allotted to him for treating diseases of the eye in the vast Allgemeines Krankenhaus in Vienna. His writings, and those of Himly and Langenbeck of Göttingen and Schmidt of Vienna, were scarcely known in England or America, except perhaps by the few who could read the German language.

It was the declared purpose of those who labored at the London Eye Infirmary to bring back ophthalmology into the field of legitimate medicine and surgery in England. In the words of Samuel Cooper, as quoted by Travers, it was believed then as it is today, that "no one except the thorough surgeon can make the complete oculist."

STATE OF OPHTHALMOLOGY IN AMERICA ON THE RETURN OF THE FOUNDERS

The College of Physicians and Surgeons of New York that had graduated Delafield and Rodgers stood not alone in its neglect of diseases of the organs of vision in its curriculum. It was a matter of disregard in all the colleges then existing in this country. George Frick, far away in Vienna, "felt deeply the dearth of knowledge of diseases of the eye in America, and set himself to work to so qualify himself that he might return to his home and give some enlightenment and scientific uplift to a neglected department of medicine." Similar thoughts passed through the minds of Delafield and Rodgers during their sojourn in London. Returning to New York in 1818, they made a survey of the condition of persons suffering from diseases of the eye in this city. Surely no previous investigation had ever been made along these lines in this country. It had been noted that comparatively few such persons applied for relief at the public institutions. At the same time it was realized that a great many people were constantly suffering with diseases of the eye, for whose relief there was no provision. It was considered that the poor so afflicted could hardly be counted as fit objects of the New York Hospital, the function of which was to treat diseases of a more formidable nature that were actually dangerous to life. From the medical attendant at the City Almshouse at Bellevue, it was learned that a considerable number of paupers were annually received and supported there, incapacitated by the condition of their eyes. It was further known that a still more numerous and useful class, consisting of trades-

men and mechanics, of women with small incomes and of various others who had just enough to support themselves in health, could not afford to pay for good advice when afflicted with diseases of the eye, and yet they were too proud and independent to apply to a hospital or dispensary. It was thought that to an institution devoted solely to the treatment of diseases of the eye self-respecting people in moderate circumstances would go without hesitation.

Independent of these humanitarian considerations, no institution existed in America in which a sufficient number of patients suffering from diseases of the eye could be seen to afford adequate experience in the treatment of these diseases or to give an opportunity for teaching them. It was known already that there is a wide variety of diseases of the eye that are so similar that it requires a large experience to distinguish them. It was also observed that few operations on the eye were ever done in a general hospital, so that it was beyond the reach of the general surgeon to acquire dexterity in operating on this organ. The minds of the medical profession in this country, always sensitive to happenings in the mother country, were being directed to the subject of diseases of the eye by the work of Scarpa, which had been translated into English, and by the writings of Saunders, published posthumously, which found their way into the private libraries of the medical profession.

THE FOUNDING OF THE INFIRMARY AS A PRIVATE INSTITUTION

The founders first conceived the idea of organizing an infirmary in New York for curing diseases of the eye while studying at the London Eye Infirmary, in 1816. To quote their own words, "the primary object in establishing the Infirmary was to contribute towards the relief of the poor who by a diseased state of one of the most important organs of the human body are deprived of the means of gaining a livelihood." It was not until four years after the conception of the idea that they ventured to take steps to fulfil their dream. Their most urgent need was to establish themselves in practice and gain the respect of their fellow citizens and of the medical profession. At the end of two years in private practice they made bold to take up the actual task of establishing an eye infirmary in New York similar to the one in London—"for the first time in America," as was affirmed by them on many occasions. Had Delafield and Rodgers known of any other eye infirmary in this country, worthy of the name, in existence before the birth of the New York Eye Infirmary, it seems certain they would have accorded to it priority of foundation. In the undertaking they obtained the countenance and advice of Drs. Wright Post and Samuel Borrowe, distinguished medical

officers of the New York Hospital, whose names served as a guarantee to the public of the respectability of the project and as an assurance to the medical profession that medical ethics would be observed.

THE FIRST HOME

The first home of the Infirmary consisted of two small rooms on the second floor of a building at 45 Chatham Street. The owner of the building acted as superintendent, and students of the College of Physicians and Surgeons volunteered to act in turn as apothecary, a task they were well qualified to do, since physicians were still carefully taught how to compound their own prescriptions. The hours were from 12 to 1 on Mondays, Wednesdays and Fridays, and all medicines were given gratuitously. It was resolved by the founders to solicit no public assistance until first they had demonstrated the success and utility of the undertaking. Within a short time the rooms were crowded during the hours of practice, and it became apparent that the demand was too great for the efforts of two men just starting out in practice.

TRANSFORMATION OF THE INFIRMARY INTO A PUBLIC INSTITUTION

On the evening of March 9, 1821, seven months after the opening of the Infirmary, and after 436 patients had been treated, an event occurred of far-reaching importance to the Charity. Acting on the advice of older men, the founders arranged for the holding of a public meeting, the purpose of which was to devise means of perpetuating the benefits of the Infirmary. The meeting was held at the famous City Hotel that occupied a block on Broadway, between Cedar and Thames Streets, long remembered for the excellence and variety of its wine cellar. The aged William Few, a veteran of the Revolution, was called to the chair, and James Eastburn acted as secretary. It was a distinguished gathering judged by the names that have been handed down through the mist of years. Delafield read a report on "The History and Progress of the New York Eye Infirmary." In that paper the audience was told of persons perfectly blind who were restored to sight and of others who were relieved after they had tried countless unavailing remedies and had ceased to hope until they learned of the Infirmary. It happened to the surgeons that on the same day they saw people with eyes irretrievably lost from diseases in childhood, and at the same time children were brought in suffering with the same diseases in a state admitting of early cure. Three particular cases were cited for the information of the assembly of persons "born blind" who had been actually restored to sight and given the use and enjoyment of a special sense hitherto unknown to them. These were in all probability the first cases of con-

genital cataract successfully operated on in the New World, as the operation then in vogue of couching was not applicable in this form of cataract. Saunders, shortly before his death, introduced the method of needling for congenital cataract on the principle that free lens matter dissolves in the aqueous.

As a result of the meeting, a committee was appointed to take over the affairs of the Eye Infirmary and to raise funds for its maintenance. On motion of Philip Hone, afterward the "gentleman mayor" of New York, seconded by Dr. David Hosack, a vote of thanks was tendered to the founders for the gratuitous services they had rendered, and Mr. Jennings, the proprietor of the hotel, was thanked for the free use of the room. No meeting of a medical nature in New York would have been considered complete without the presence of David Hosack, called the most eminent physician of America. It was he who attended Alexander Hamilton after the duel with Aaron Burr. His name brings one close to the death-chamber of Washington, for Hosack's friend, Dr. George Augustus Dick, who was said to have first interested him in botany, was still living as the last survivor of the three consultants in Washington's last illness, who were accused by an indignant nation of having bled the great man to death. Doubtless the youthful founders, neither one of whom was much over 27 years of age, as they wended their way home through the dimly lighted streets, experienced a feeling of just pride and exultation.

THE FIRST BY-LAWS

At a meeting held April 21, 1821, to a committee composed of Colonel William Few, David B. Ogden and Dr. Delafield, the task was given of drawing up suitable by-laws. It is interesting to note that one of the members, William Few, had been a member of the convention that framed the Constitution of the United States. No wonder that the original by-laws, carefully worded, concise and comprehensive, and beautifully written, in an age when penmanship was an art, bear silent witness to the seriousness with which these gentlemen went about their task. It was provided that a donation of $40 constituted one a life governor and entitled him to the privilege of having at all times two persons under treatment at the Infirmary, free of charge. The governors had the right to vote for and to become directors, of whom there should be seventeen. The organization was to be known in its corporate capacity as "The Society of the New York Eye Infirmary," a quaint appelation that unhappily has fallen into disuse. Provision was made for the appointment of two surgeons and two consulting surgeons, who should receive commissions under the seal of the Society and hold their offices under good behavior. Strict rules were laid down for dealing

with directors or surgeons who should prove unfit for their duties or guilty of misdemeanors themselves. The names of the first officers of the Infirmary, which is the fourth oldest hospital in New York City, and the oldest hospital of its kind in the United States, are now recorded "with honorable praise":

William Few, president
Henry I. Wyckoff, first vice president
John Delafield, Jr., treasurer
James I. Jones, secretary

Directors

Nathaniel Richards	Jeromus Johnson
Benjamin L. Swan	Isaac Collins
William Howard	Cornelius Heyer
Henry Brevoort, Jr.	Henry Rankin
John Jones	Benjamin Strong
William Howell	Samuel F. Lambert
James Boggs	Edward F. Laight
Isaac Pierson	Gideon Lee

Surgeons

Edward Delafield	John Kearney Rodgers

Consulting Surgeons

Wright Post	Samuel Borrowe

The entire surgical staff of the Infirmary was on active duty at the New York Hospital. A glance at the names of the directors reveals that a number of them were among the governors of that institution. Thus the New York Eye Infirmary may be considered as nothing more or less than an offshoot of the New York Hospital. In like manner, the London Eye Infirmary, through Saunders, Travers, Lawrence and Cooper, sprang from the great general hospitals of St. Thomas's and Guy's. It was unfortunate that a cleavage developed in the course of time between the daughter infirmaries and the mother hospitals.

THE SECOND HOME

Some time in the year 1822, the Infirmary was moved from its birth place to larger quarters at 1 Murray Street, at the corner of Broadway. An apothecary was appointed to take over the duties that until then were performed by medical students. He was to have charge of the electrical apparatus and to apply it when directed. It was a machine, a mere toy in the light of today, that was supposed to possess wonderful powers in the treatment of "paralysis, blindness, deafness, and apparently any disease that had hitherto been thought incurable." He was to have in his care all surgical instruments, and was never to suffer them to be lent or used by any person except the surgeons of the Infirmary. An important office of his, when directed, was to perform the operations

of bleeding and cupping and the application of leeches. He was to see that all patients were kept away from the apothecary shop, a rather difficult task because the laity was in the habit of consulting the apothecary for all ordinary ailments, reserving only serious illnesses for the physician. A further glimpse into the activities is afforded by the instructions to the porter, who was to be present with the patients in the anteroom during the hours of prescription, remaining with them and arranging them for treatment in the order in which they came.

GRANTING OF THE CHARTER

By an act of the legislature of the state of New York dated March 22, 1822, the Charity was incorporated under the title of "The New York Eye Infirmary." By this name it was known for forty-two years, until in 1864 the name was changed to its present one. In memorializing the legislature, it was pointed out that the advantages accruing to the public from the London Eye Infirmary had become so apparent that similar infirmaries had been formed in all the principal cities of Great Britain, and that the celebrity of the London institution had reached to the remotest corners of the globe and its blessing diffused wherever the English language was known. It was stated that in ten years more progress had been made in the diagnosis and treatment of ocular diseases in England than had been made in the previous fifty years. It was related that reports of the successful treatments and operations at the New York Eye Infirmary had already spread to all parts of the state and that the existence of such an institution had come to be looked on as a necessity to the public welfare.

EARLY STRUGGLES OF THE INFIRMARY

"What good gift have you, my brother, but it
Came from search and strife and loving sacrifice."

The rooms at 1 Murray Street were from the beginning inadequate for the purpose of the Infirmary. There were no beds. Patients on whom the most serious operations had been performed were compelled to go back to their homes at once. That operations failed and that eyes were lost, the surgeons attributed to dirt, overcrowding and complete lack of care in the lodgings to which the patients had to be returned. What the surgeons expressed themselves as needing were wards with "fresh air, good ventilation, regulated heat and good nursing." Of hygiene, they had excellent ideas, although they had no thought of surgical cleanliness and the invasion of micro-organisms. A petition was sent to the Corporation of the City of New York for rooms, but none were found available. A similar request was made of the governors of the New York Hospital, but they lent a deaf ear. An

effort was made to arrange with the New York Dispensary, founded in 1795, for the erection of a building to be occupied by both, but the proposition failed. The finances of the infant Infirmary were chronically low, and it was necessary for the landlord to be indulgent. The founders were untiring and resourceful. They sought the aid of the clergy. Sermons were preached and collections were taken up in various churches in behalf of the Charity. Among those who rendered great service was the Rev. J. M. Wainwright, rector of the original Grace Church, at the corner of Rector Street and Broadway, and long a member of the board of directors of the Infirmary. It was said that no truer christian gentleman ever ascended a New York pulpit than he. So desperate was the Infirmary, that there is mention in the records that a person was hired for the purpose of soliciting funds. The undertaking was not looked on with favor in all quarters. It was argued that charitable institutions are injurious to the public and that they create an evil to which they claim to apply a remedy and offer a premium on idleness. To which the sponsors of the Infirmary replied that these objections could never be laid against the Infirmary, but that on the contrary, in restoring persons to sight it would add to the number of industrious citizens and save many from the necessity of subsisting on the public bounty. The year 1822 was a particularly critical one. Not only was there financial trouble, but yellow fever broke out in the city, and all that could of the terror stricken inhabitants fled to Greenwich Village. The doors of the Infirmary were closed for three months during the epidemic, this being the only time in its long history that such a step has been found necessary.

AN ACT OF THE LEGISLATURE FOR THE RELIEF OF THE INFIRMARY

The board of directors, despairing of raising sufficient funds locally for obtaining a building, petitioned the legislature. They had an example to go by in the New York Hospital to which the legislature had some years previously granted a sum out of the excise tax for the purpose of erecting a building on the grounds of the hospital for housing the insane. The petitioners pointed out that the example of the New York Eye Infirmary had been followed by the establishment of a similar institution in Boston and one in Philadelphia. The one referred to in Boston was the Massachusetts Charitable Eye and Ear Infirmary, founded by Edward Reynolds and John Jeffries, at the suggestion of Delafield and Rodgers, and the one in Philadelphia was a forerunner of the present Wills Hospital, which was not established until ten years later. In the memorial, a violent ophthalmia in the northern and western counties of New York was described that had been raging for

several years, and that had resulted in the partial or total blindness of innumerable people. Many patients from this area made the tedious journey down to the Infirmary for treatment. Some of them were already hopelessly blind. The surgeons were pained at having to turn away many of the victims of this epidemic and others, whom they might have helped, because of lack of wards to accommodate them during the period necessary for their treatment. The legislature was informed that many diseases of the eye, while easily curable in the early stages, are intractable at a later period. An Act of Relief was passed April 1, 1824, granting to the Infirmary the sum of $1,000 annually over a period not exceeding two years, for the express purpose of providing itself with a building adequate to its purposes. This grant was renewed for many years and was long the chief support of the Infirmary.

THE THIRD HOME

With this fund in hand the Infirmary was able to lease from the governors of the New York Hospital a portion of the building that had been occupied as a lunatic asylum, the asylum having been removed to Bloomingdale a year or two previously. As already stated, this building was located within the spacious grounds of the hospital, and was known as 139 Duane Street. The new quarters consisted of a kitchen and one room on the first floor and the entire second floor, with the use of the yard. Within these walls, already inured to the sight of human suffering, there were gathered together the first indoor patients of the Infirmary, under the protection of a great general hospital. Patients were boarded by the superintendent at $2.50 a week, if they were able to pay, otherwise they were cared for free of charge as the funds permitted. A resolution was passed requiring each patient from the country to give a bond that he would not be chargeable to the city on being discharged from the ward. Immediately there was noted an improvement in the result of the operations. Fewer eyes were lost than formerly. People hearing of the wonders done came from afar to be operated on, many of whom hitherto had had little faith in the ability of surgeons to help them. From 1824 to 1826, the Infirmary was sheltered within the confines of the New York Hospital. One cannot but ponder that had that venerable institution created a permanent place for the infant Infirmary, in its scheme of relieving human suffering, other institutions in distant cities might have followed the example, with the result that American ophthalmology would have been spared the process it is today undergoing of "reaffiliation" with general medicine and surgery. However, ophthalmology needed to be separate and apart for a season, free from the restrictions that would have been thrown

about it in hospitals devoted to larger issues. These hundred years ophthalmologists have solved many problems and on the whole have rendered a good account.

TEACHING

The Infirmary has always had a strong bent toward teaching and training young men in a practical way, a trait that it may be said to have inherited, along with its esprit de corps, from the New York Hospital which has always been noted for its educational work. Rodgers trained Agnew, and Delafield trained Noyes, under whose influence came Weeks. One lives in the knowledge gained from others. Neither of the founders wrote much. They were far too busy with other matters, and besides their younger years were passed in a period when there were

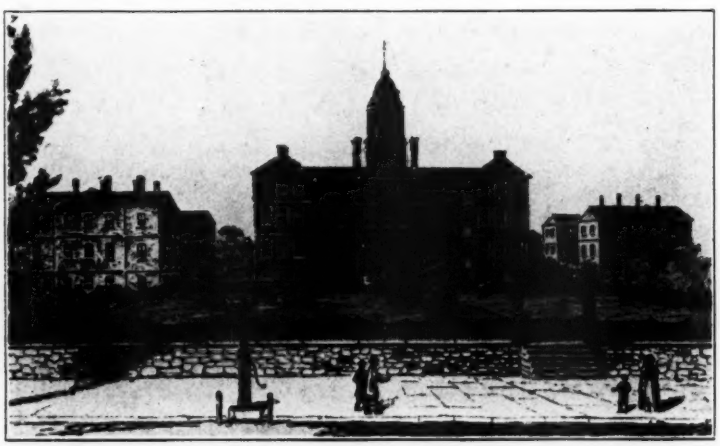

Fig. 1.—The New York Hospital in 1808. The Infirmary occupied a portion of the second building on the left from 1824 to 1826.

few medical journals. Contributions to the literature usually took the form of treatises. So greatly did the medical profession stand in need of instruction in diseases of the eye that the legislature took cognizance of the fact and stipulated in the Act of Relief that at least one medical student from each county of the state should be admitted to witness the practice and surgery at the Infirmary at all times, free of charge, provided he first presented a certificate from the president of his county medical society. After the Revolution, the medical profession in America experienced a great impetus. During that prolonged struggle physicians were called from isolated spots in the country and thrown together in the army to their mutual benefit. Many of them witnessed the excellent surgery performed by the French and British in the foreign armies. At the end of the war, county societies sprang up and became very important, but it was twenty-seven years after the birth

of the Infirmary before the American Medical Association was organized. To inform the medical profession of the terms of the Act of Relief, the board of directors caused advertisements to be placed in all the papers of the state and notices to be posted at the College of Physicians and Surgeons. There was now in the English-speaking world a hospital, perhaps the only one, wherein students of medicine had a legal right "to walk the wards." Up to this time medical schools and hospitals were quite distinct. Students were admitted to the latter only by the sufference of the governors, and we are told "they were not always welcome." During the winter of 1823, mention is modestly made in the records that one of the surgeons (presumably Edward Delafield) was beginning a series of lectures at the Infirmary on diseases of the eye, which were to be continued each succeeding winter during the session of the College of Physicians and Surgeons. It was from this source that most of the early students were drawn. The pupils who attended the lectures were granted the privilege of witnessing the practice and surgery at the same time. These lectures, in all likelihood, represent the first systematic effort at teaching ophthalmology in this country. They were probably didactic, as clinical lectures had not been adopted in medical schools, and before they should be it was held against them that they are "misleading, ineffectual and superficial." By the end of the third year after the foundation, thirty-seven students had received instruction and were pronounced capable of practicing this special branch of medicine in the circles to which they would return. Nothing could have been farther from the minds of the founders than to turn out "specialists." In fact, specialization was not looked on with approval in the first half of the nineteenth century. It was thought that every physician should be big enough to gain a practical knowledge of all branches of his profession. The prevailing opinion was that a useful knowledge of diseases of the eye could be obtained in a few months, and with comparatively little labor. The diseases were said to be nearly all open to inspection, and their results could almost always be predicted even if they could not be prevented. It was fully forty years after the foundation of the Infirmary before ophthalmologists began to prescribe glasses as a therapeutic measure, the matter of refraction being left undisturbed in the hands of the opticians.

NATURE OF THE CLINICAL MATERIAL

In the first annual report issued in January, 1822, covering the first sixteen months, it is announced that 1,120 patients had taken advantage of the Charity. Of this number the surgeons made the following recapitulation, which is most valuable as giving an idea of the nature

and extent of maladies of the eye at the time of the foundation of the oldest eye infirmary in the Western Hemisphere:

Inflammation of the conjunctiva	276
Inflammation of the conjunctiva with purulent discharge (adult)	24
Inflammation of the conjunctiva with purulent discharge (infants)	15
Strumous inflammation of the conjunctiva	124
Strumous inflammation of the conjunctiva with pustules	48
Strumous inflammation of the conjunctiva with ulcers	7
Chronic inflammation of the conjunctiva with thickened eyelids	10
Chronic inflammation of the conjunctiva with vascular cornea	15
Excrescences of the conjunctiva	4
Pterygium	4
Opacity of the cornea	60
Staphyloma	6
Inflammation of the iris and its consequences	76
Cataract *	51
Amaurosis	113
Strabismus	5
Paralysis of the upper eyelid	8
Tinea ciliaris	83
Lippitudo	98
Inversion of the eyelids	3
Tumors of the eyelids	9
Diseases of the lacrimal passages	26
Exophthalmia	4
Dislocated lens	1
Wounds and other injuries of the eye	19
Anomalous diseases	22
Total	1,120

* "Of the cases of cataract three were persons born blind from this cause and in each sight was restored by operation."

Imagine 76 cases of inflammation of the iris and its consequences, 60 cases of opacity of the cornea and 113 cases of amaurosis in a little over 1,000 patients! What a heavy toll on sight was exacted by disease then as compared with today! Of operations, there were relatively few outside of couching for cataracts. This was described as singularly free from pain, if properly managed, and not nearly so distressing as the presence of a foreign body between the globe and the lids. The Creator was thanked for His beneficence in rendering the eyeball so nearly insensible, enabling it to undergo those operations that are so frequently necessary for the recovery of sight.

ESTABLISHMENT OF THE AURAL DEPARTMENT

During the year 1823, steps were taken to ascertain the desirability and legality of forming a new branch for the treatment of diseases of the ear. As nothing seemed to stand in the way, the founders began to treat aural conditions without more ado. They were surprised at how well many of the diseases responded to treatment. It was not until April 13, 1825, that a third surgeon was appointed, who was to have exclusive charge of the ear in all subjects of the Infirmary. This first aurist is recalled in a rather bad aura. He absented himself

without leave, and as he paid no attention to communications he was presumed to have resigned. In his place another aurist was appointed, who did no better. He also neglected his duty and was considered to have resigned.

SEAL OF THE INFIRMARY

At a meeting held early in 1824, the treasurer, John Delafield, Jr., a brother of Edward Delafield, was instructed to see to the preparation of a seal. It was he who some years later was on the committee, with John

Fig. 2.—The seal of the New York Eye and Ear Infirmary, adopted July 2, 1824.

Trumbull, William Dunlap and Philip Hone, that passed on the likeness of the famous statue in marble of Hamilton that was placed in the Merchants' Exchange in New York. On July 2, 1824, a seal was produced at a meeting of the directors which was adopted as the seal of the infirmary. It depicts the Great Physician restoring to sight a man who was "born blind," prior to which event "it was not heard that any man opened the eyes of one that was born blind." The seal stands for all time as a symbol of the religious background and piety of the founders.

At this same meeting a treatise on diseases of the eye, being a present from its author, George Frick, was laid on the table. The

secretary was instructed to convey the thanks of the board to Dr. Frick. This was the first book on ophthalmology written in America. It was largely a compilation of the lectures that the author heard Beer deliver in Vienna. It proved so popular that an edition was brought out in England. In this way Frick rendered the English-speaking ophthalmologists a great service by opening to them the teachings of the Vienna School of Ophthalmology. He died in Dresden in 1870, aged 77 years. His deeds deserve a wider appreciation.

Fig. 3.—Colonel William Few. (From an oil painting.)

BIOGRAPHICAL SKETCHES

WILLIAM FEW

Of all the illustrious laymen, without whose help the Infirmary could never have been started on its long career, no figure seems to arouse such a personal interest as that of the first president, Colonel William Few. He was born in Baltimore in 1748, not far short of two hundred years ago. His ancestor, William Few, came over with William Penn. Colonel Few was reared in the South. In his youth he had an insatiable thirst for knowledge, educating himself by reading whatever came in his way and by attending the county court. He earned his title in the revolutionary army in engagements against the British and Indians. Among many high offices he was a member of the Continental Congress, and was one of the first United States senators from Georgia. He

removed to New York in 1799, of which city he became mayor, at a time when the mayor was not elected by the people but was appointed by the governor. How this old warrior and statesman ever became interested in the establishment of an eye hospital may never be known. He presided over the destinies of the Infirmary for eight years, closing his long and eventful life in 1828. *Finis coronat opus.*

JOHN KEARNEY RODGERS

John Kearney Rodgers was born in New York City, in 1793, of Scotch ancestry. He was a son of Dr. John R. B. Rodgers, an attend-

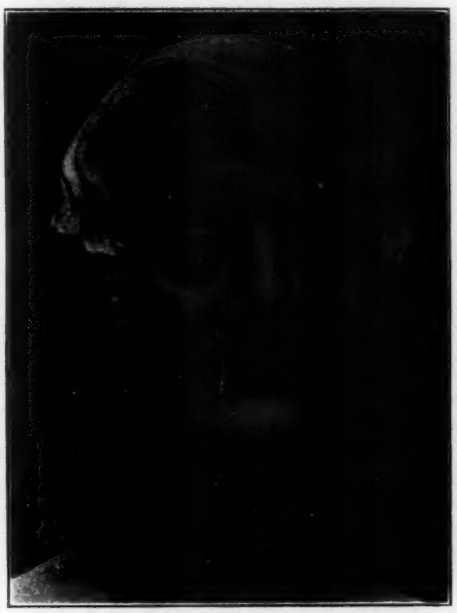

Fig. 4.—John Kearney Rodgers.

ing physician at the New York Hospital, and grandson of the Rev. John Rodgers, pastor of the Wall Street Presbyterian Church. He entered the College of Physicians and Surgeons by way of Princeton. For some reason he was not in favor with the president of Princeton, Dr. John Stanhope Smith, who once remarked to him that he would never distinguish himself, to which he replied, smarting under the taunt, "the world shall see, sir." Dr. Rodgers related this incident many times, saying that this was the first stimulus his ambition ever received. In London, Sir Astley Cooper took a very different view of him, singling him out among the 400 students who attended his lectures, as a man of the greatest promise. One day in May, 1817, he witnessed that famous surgeon ligate the aorta, considered up to that hour the greatest feat

in the annals of surgery. Before leaving London, he passed the examination and received the license of the Royal College of Surgeons. Twenty-eight years later Rodgers emulated the example of the great master, in the arena of the New York Hospital, by tying the innominate artery within the scalini muscles, an operation believed never to have been successfully performed before. As an ophthalmic surgeon at the Infirmary, he is spoken of as having been "quick, skilful and brilliant," in which respect he differed from his master, Sir Astley Cooper, who said of himself that for the operation of cataract he was unfitted by nature. He thought too much! Rodgers was noted for his ability and

Fig. 5.—Edward Delafield.

integrity, and was called by the laity "the good physician." A whole city was thrown into mourning at his untimely death in the zenith of his reputation in 1851. That was the year of the invention of the ophthalmoscope, which as von Graefe said "crowned the head of Helmholtz with the laurel of immortality." Rodgers died in sight of the promised land of ophthalmology into which Delafield was to enter and receive great honors. Between the two there existed a life-long friendship of rare intimacy and perfect accord.

EDWARD DELAFIELD

Edward Delafield was born in New York City in 1794, coming of a family of high social position and great wealth. His classic education

was received at Yale. He became a most important personage in the medical world of New York. He was long president of the College of Physicians and Surgeons, of which institution he was emeritus professor of obstetrics and diseases of children. In his day many physicians distinguished themselves in more than one branch of medicine. He was unanimously elected the first president of the American Ophthalmological Society, on its organization in 1864, in recognition of his valuable services in assisting in the elevation of ophthalmology to an honorable and important place in the minds of the profession. He lived to see the "oculist" recognized, in the expression of Herman Knapp, "as a physician and surgeon who wants to gain time for perfecting himself in the knowledge and treatment of diseases of the organ of the highest sense more thoroughly than it would be possible if he remained in general practice." He was a person of decided views, detesting tobacco in any form. How he stood on the subject of alcohol is not recorded, but he probably had notions different from those of Saunders, Moorfields' founder, who two hours after having been induced to drink three glasses of wine was stricken dead with apoplexy. A contemporary account describes him as being 5 feet 10 inches in height and weighing 150 pounds. In spite of his arduous duties and the many epidemics through which he ministered, his life was spared until 1875, fifty-five years after the foundation of the Infirmary. At its semicentenary celebration in 1870, he stood a tragic figure, the sole survivor of the original board of directors and surgical staff. He often related how the Infirmary came to be founded, and at his death he remembered it with a handsome bequest.

We take just pride in the founders, in the knowledge that they were men of extraordinarily high attainments and proud pedigrees. Traditions of their courtly manners and knightly deeds inspire us today. It is a great privilege to be able to link their names with the birth of the Infirmary. But Delafield and Rodgers were more than cofounders of one institution. Edward Reynolds acclaimed them in 1850, and no one knew better than he whereof he affirmed, as the fathers of American ophthalmology.

MYASTHENIA GRAVIS

NEW DIAGNOSTIC EYE FINDINGS, WITH POSSIBLE PATHOLOGIC SIGNIFICANCE

S. V. ABRAHAM, M.D.
LOS ANGELES

Myasthenia gravis was first described by Wilkes in 1877. Since then about 300 cases have been reported and several excellent reviews have appeared (Buzzard,[1] Campbell and Bramwell,[2] Keschner and Strauss,[3] Jaensch,[4] Marinesco, Sager and Kreindler [5] and others). It is intended here to recapitulate briefly the more important observations and theories, to discuss some new eye findings in the light of these theories and to consider these new findings as possible diagnostic aids.

The condition can probably be most briefly defined as a symptom complex characterized by sudden, intermittent and progressive clinical weakness of a muscle or muscle group, often terminating in death and leaving almost no pathologic changes despite the marked symptoms during life. No race is immune to this condition. According to Campbell and Bramwell, no case is on record in which more than one member of a family has been affected. Noyes,[6] however, reported a sister and father of one patient to be similarly afflicted. The condition is perhaps more frequent in females than males, with an earlier age incidence in the former. The age of onset varies from early childhood to senility, but is most commonly between 20 and 50 years. The condition may occur after pregnancy or after an acute infection, but more often seems unrelated to any other condition. Its onset may be sudden or gradual.

Submitted for publication, Nov. 24, 1931.

Presented before the Los Angeles Society of Neurology and Psychiatry, Nov. 25, 1931.

Work done while instructor in the Department of Ophthalmology, University of Chicago, Dr. E. V. L. Brown, Director.

1. Buzzard, E. F.: The Clinical History and Post-Mortem Examination of Five Cases of Myasthenia Gravis, Brain **28**:438, 1905.
2. Campbell, H., and Bramwell, E.: Myasthenia Gravis, Brain **23**:277, 1900.
3. Keschner, M., and Strauss, I.: Myasthenia Gravis, Arch. Neurol. & Psychiat. **17**:337 (March) 1927.
4. Jaensch, P. A.: Disturbances of the Ocular Muscles in Myasthenia, Ztschr. f. Augenh. **71**:292, 1930.
5. Marinesco, G.; Sager, O., and Kreindler, A.: The Pathogenesis of Myasthenia, Ztschr. f. klin. Med. **113**:404, 1930.
6. Noyes, A. P.: A Case of Myasthenia Gravis, with Certain Unusual Features, Rhode Island M. J. **13**:52, 1930.

There may be periods of remissions lasting for many years of apparent good health. The condition may be permanently arrested or may grow slowly or rapidly worse, involving both smooth and skeletal muscles. The patient becomes bedridden, incapable of talking or swallowing and may finally die of asphyxiation or of aspiration pneumonia. Those muscles used most are frequently first involved, such as the muscles of mastication, the eye muscles, the fingers (of a pianist), etc.

According to Starr,[7] ptosis or diplopia is the first symptom in 40 per cent of the cases. Dana [8] reported diplopia as the earliest symptom in 50 per cent of the cases. Klar,[9] quoting Bielschowsky, stated that ptosis is present some time in the course of the disease in 80 per cent of the cases. The latter agreed with Uhtoff, quoted by Posey.[10] There is no doubt that eye findings are present early in a very large percentage of cases. In many more cases the eye symptoms, while not the first symptoms in matter of time, are often the first for which a doctor is consulted.

There is a ptosis of one or both lids which varies from time to time, usually being least marked in the morning or after rest. Sometimes the ptosis is more marked on one side and sometimes on the other. It can usually be brought on or increased by exercise for a short time. This phenomenon of making apparent or increasing the severity of the symptoms by exercise is one of the characteristics of the disease. Emotional upsets do the same thing. An unstable vasomotor mechanism is usually a concomitant finding, as is evidenced by the rapid flushing, Quincke's edema and urticaria frequently present. Limitation of the ocular movement is frequent and subject to great daily variation. Even in the severe cases the pupillary reflexes are usually normal. Occasional sluggish reflexes are reported, but are so infrequent as to be of no importance (Posey). Except in extreme cases with prostration, changes in accommodation are also rare. When they are found, there is a gradual recession of the near point on repeated tests. Posey did not believe the nystagmus reported to be true nystagmic movements.

Some unusual findings recorded in the literature, not easily explained on the usual assumption of a muscle weakness, are lid lag, champing of the jaw (Buzzard), biting of the tongue, "a smile that would not come off" and difficulty in reopening a flexed hand (Dana). Continuation of the muscle response after the stage of failure to respond to the faradic current has been reached (reaction of Jolly) by (1) a

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7. Starr, M. A.: Myasthenia Gravis, J. Nerv. & Ment. Dis. **39**:721, 1912.

8. Dana, C. L.: Myasthenia Gravis, J. A. M. A. **78**:261 (Jan. 28) 1922.

9. Klar, J.: Eye Symptoms in a Case of Myasthenia with Thymus Tumor, Klin. Monatsbl. f. Augenh. **85**:224, 1930.

10. Posey, W. C.: Myasthenia Gravis, J. A. M. A. **77**:1007 (Sept. 24) 1921.

change in the polarity, (2) a change in the strength of the current, (3) a change to galvanism and (4) voluntary effort (Campbell and Bramwell, Keschner and Strauss) also does not clearly support an assumption of a muscle weakness. Herzog [11] has reported a case with increased tonus bordering on tetany.

The data herewith presented are also not in keeping with the idea of a muscle weakness. However, for a clearer understanding of these data it is necessary to refer first to a method of testing for heterophorias used by me for several years and recently described.[12] In connection with this test the ordinary Maddox rod test plays an important part, but the test is of prime importance because of the production of "innervational equilibrium" by a continuous method of taking ductions. In this method a Risley prism (30 prism diopter capacity) is placed before one eye and rotated base up and down alternately with both eyes open and fixed on a small (from one-half to three-fourths inch [1.2 to 1.9 cm.]) target 20 feet (6.1 meters) away. The patient sees alternately "one," "two," "one," "two," etc., and the prism power is very gradually increased and decreased, a note being made of the amount of prism base up (or down) at which the patient first sees "two" after having seen "one." This can be repeated any number of times and readings taken (chart 1). At any time during the foregoing procedure, as well as at the beginning, the Maddox rod can be slipped before either eye, producing a horizontal streak. The amount of "hyperphoria" present at that moment can then be measured by the amount of prism, base up or down, needed to make the streak pass through the center of the light. The use of the Maddox rod in heterophoria tests depends on the principle of suspending fusion by the marked distortion of one image. The heterophoria is then manifest and is measured by prisms. The use of prisms in conjunction with the Maddox rod test is based on the optical property of prisms to refract rays of light passing through them toward their bases. This same property permits the use of prisms in duction tests. Because of this optical property and the law of projection of images, the eye being stimulated of necessity will move as long as binocular single vision is to continue, in such a way that the corresponding part of its retina is stimulated as compared with the retina of the other eye; that is, if the right eye is fixed on an object and a prism is placed before the left eye, the left eye will so move that its foveola is also stimulated by light coming from this same object. This means that

11. Herzog, F.: Ueber die myasthenische Ermüdung auf Grund der Untersuchung des Aktionsstromes der Muskeln und des histologischen Befundes, Deutsches Arch. f. klin. Med. **123**:76, 1917.

12. Abraham, S. V.: Heterophorias; I. A New Test for Vertical Phorias with Observations on Patients with Presumably Negative Histories, Arch. Ophth. **5**:766 (May) 1931.

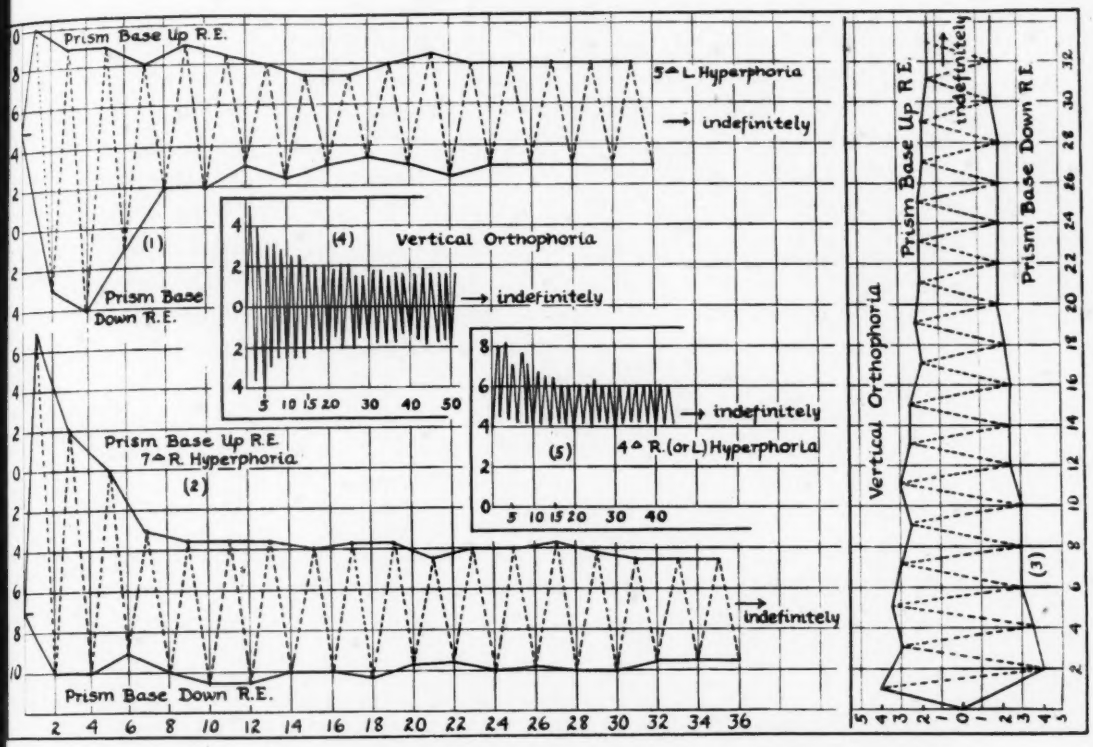

Chart 1.—(1) A case of 5 prism diopters of left hyperphoria rarely shows any ability to overcome prism base down before the right eye after the first few readings. Usually innervational equilibrium is established early, as shown in the graph. (2) A case of 7 prism diopters of right hyperphoria rarely shows any ability to overcome prism base up before the right eye after the first few readings, usually showing innervational equilibrium as indicated in the graph. Here the initial reading is prism base up before the right eye and is relatively high, but it drops rapidly to minus (below zero) prism base up before the right eye. (3) This graph shows that only minor variations in the vertical ductions are present in the normal state. A case of vertical orthophoria rarely shows higher initial readings than is indicated here, and soon there is a drop to 1½ or 2 prism diopters of right and left sursumduction. The readings base up and down from the right eye are taken alternately but continuously. (4) This graph is similar to graph 3. (5) In this graph the ascending portions of the curves show the area in which the patient sees singly, while the descending portions indicate the area in which diplopia exists. The change in direction downward indicates that the patient has just seen double, while a change upward indicates that the patient has just seen singly. Note that the space between these two series of points is dependent on the hyperphoria present, that is, that the patient sees singly (following diplopia) only after the amount of prism closely approximates the amount of hyperphoria. This is true in the nonmyasthenic person whether the hyperphoria is large or small, or whether no hyperphoria is present, so that this graph can represent any assumed condition simply by being raised or lowered (varying the ordinates). When the diplopia is noted at any time in any of these graphs, the images are at least from 6 to 8 cm. apart, depending directly on the amount of prism before the eye. (In these and the following graphs the ordinates represent the amount of prisms and the abscissae represent the readings.)

as long as the fusion center is active, as long as single binocular vision can be maintained, the eye behind the prism will rotate in the direction of the apex of the prism, and the muscles contracting are those which perform this function. That this is what takes place is evident from actual observation in case 3 reported here.

If, despite the maximal fusional efforts to maintain single binocular vision, this is impossible, diplopia results. This method of measuring heterophorias is based on the production of an insurmountable diplopia in one direction while measuring any manifest deviation in the other direction. The condition that permits this is the almost complete suspension of fusional effort. The eyes then take up a position of relative rest, permitting the measurement of heterophorias. If vertical (horizontal) prisms are used to produce the diplopia, then the images are separated in space by an amount directly proportional to the amount of prism used, even though the patient had only a moment before been able to overcome almost the entire amount of prism used. This point is emphasized as indicating that, despite previous effort, the muscles immediately relax almost entirely on suspension of fusion, a condition that does not exist in the myasthenic patient, as the following data indicate.

CASE 1.—H. E., the patient, a woman, aged 37, gave the following history on April 4, 1929. Her chief complaint was extreme exhaustion for four years. Present Illness: The first symptom noted was loss of the voice while the patient was reading a paper before a medical society in 1917. Three attempts to resume the paper after short periods of rest failed, although her voice was normal by the next day. In 1918 and 1919, the patient had influenza followed by pneumonia. A gradual increase in fatigue was noticed until the summer of 1924, when the patient noticed that "things moved from left to right." After two days there was a definite diplopia, especially noticeable for distant objects. An oculist at this time told her that vision was normal, but a slight Stellwag's sign was present. The patient was seen at one of the large clinics in the country and advised to take outdoor exercise. In the fall of 1924 the patient returned to medical school. One night in November, 1924, while returning from an obstetric case after being out about sixteen hours, she noticed that she could not "pick up her right foot." This difficulty vanished by the next day.

In December, 1924, the patient had a throat infection with a temperature of 103 F. and 16,000 white blood cells and showed a degree of prostration unexplained by the severity of the obvious infection. Three weeks later she dropped her suitcase twice and could not hold up her arm to comb her hair. In April, 1925, an oculist said that she had serious paralysis of the muscles of both eyes, worse on the left, with incomplete rotation. He suggested looking for a focus of infection. A submucous resection was done and the symptoms became worse. The condition was then diagnosed postdiphtheritic paralysis and the patient was put on strychnine for three weeks. While on this treatment she again fell down, but after being helped up was able to walk home. The next day liquid regurgitated through her nose and she could not hold a glass. The condition was then diagnosed as myasthenia gravis. It grew irregularly worse despite rest, suprarenal substance, thyroid, sodium cacodylate and change of climate. The most severe relapse

occurred in November, 1927, following a cold. On Dec. 17, 1927, the patient was started on Rosenow's encephalitic serum, with improvement. In February, 1928, she began taking an autogenous vaccine made from her nasal secretion. Improvement continued until December, 1928, when she had influenza and another marked set-back.

Past History: The patient had had whooping cough, measles, chickenpox and mumps in childhood. There was no history of scarlet fever, diphtheria or typhoid. Infectious diseases had included pneumonia at age of 12 and double otitis media and quinsy while the patient was in high school and college. She had always been subject to acute infections of the upper respiratory tract. The family history was unimportant.

Physical examination revealed a white, obese woman of 37, propped up in bed. The left side of the face was less expressive than the right, and the right corner of the mouth drooped. There was equal bilateral ptosis of the upper lids. The pupils were round and reacted well to light and in accommodation. There was definite

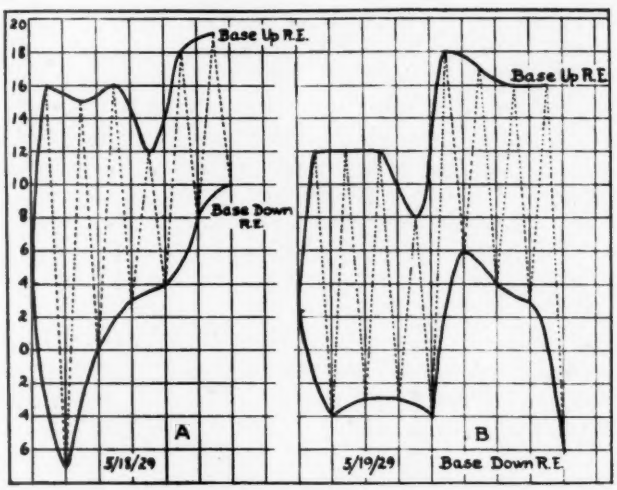

Chart 2 (case 1).—Consecutive readings (dotted line), as the left and right sursumductions are measured alternately. *A*, at the onset, on May 18, 1929, the patient had for distance (20 feet) 4 prism diopters of left hyperphoria, 8 prism diopters of esophoria and 7 degrees of plus cyclophoria. *B*, the next day (May 19), before ductions were taken, the patient showed 2½ prism diopters of left hyperphoria, from 2 to 4 prism diopters of exophoria and 5 degrees of plus cyclophoria. (Reference to the findings in the horizontal meridian as well as to the cyclophorias and cycloductions are almost completely omitted from this article, chiefly for the sake of brevity. However, these findings, while similar to those given in these and the following graphs, are not nearly so strikingly different from the normal as are the data for the vertical meridians.) After ductions were taken, the patient required 9 prism diopters base down before the left eye for fusion of images.

limitation of the ocular movements of each eye, especially on looking to the left. There was no nystagmus. The thyroid was not palpable. There was a lump in the postcervical triangle on the right side. Abdominal reflexes were not elicited. Knee and ankle jerks were equal and brisk. The Babinski reflex was absent. The neurologic summary stated: "The disturbance in the muscular activity the patient

complains of is typical of myasthenia gravis. The heart, diaphragm, chest and abdominal musculature seem to participate in the same disorder. The arms are weaker at present than the legs, but even the remarkably strong calf muscles show the typical easy fatigue after some powerful movements. In contrast to the muscular weakness, the reflexes are lively. There are no atrophies, no pyramidal and no sensory symptoms. It seems important that she has noticed that she can overcome a sudden muscular exhaustion by sugar or alcohol."

The following eye history and results of examination were obtained. May 18, 1929: The patient stated that at the onset of definite symptoms (four or five years before) there was a vertical diplopia, transient at first and later almost constant after the first hour in the morning. Later the patient noticed a horizontal diplopia also. The left cornea was definitely higher than the right. On maximum left abduction from 6 to 7 mm. of sclera remained exposed on the temporal side of the left eye. On maximum abduction from 5 to 6 mm. of sclera remained exposed on the temporal side of the right eye. On maximum downward rotation the left lower lid margin passed 2.5 mm. below the lower border of the left pupil. The right lower lid passed along the lower border of the right pupil. Other ocular movements were normal. The pupils were normal in size, shape and reactions. Vision was 1.2 in the right eye and 1.2-4 in the left. Accommodation was normal. The near point was 5.5 inches in each eye. Phoria tests at 20 feet showed: on May 18, 8 prism diopters of esophoria, 4 prism diopters of left hyperphoria and 7 degrees of plus cyclophoria; on May 19, 4 prism diopters of exophoria, 2.5 prism diopters of left hyperphoria and 5 degrees of cyclophoria.

The ductions on these dates are shown in chart 2. The patient showed good type III fusion with 9 prism diopters, base down, in the left eye, but tended to suppress the left image if no prism was used. The candle test indicated mild paresis of the left inferior rectus.

Because of the continual, consistently increased "duction" as measured by prisms, base in and base up, in the right eye and because of the relatively strong cycloductions, I made the following note in the record: "I do not feel that a muscle weakness is indicated by the eye condition."

Comment.—This was the second patient with myasthenia gravis examined by me. The first case was seen about ten days before and is given here as case 2.

CASE 2.—In O. T., the patient, a man, aged 50, the history was obtained on May 1, 1929. Present Illness: The patient complained of general weakness, starting with weakness of the lower jaw fourteen months before (August, 1928). He could not hold the jaw up or raise the arms well. He had difficulty in swallowing and speaking. Diplopia had been present since Christmas, 1928. Blurred distant vision and difficulty at near work had been noted since September, 1928. The condition had become progressively worse. The past history was essentially unimportant.

Physical examination revealed a well developed and well nourished white man who was not acutely ill. Positive findings were as follows: There was an unusual facial expression. The whole face was drawn slightly to the left. Complete ptosis of the right lid was present. The pupils were equal and round and reacted normally to light and in accommodation. External ocular movements were normal except for paresis of the right external rectus. The patient could not smile and he had difficulty in showing his teeth. The cremasteric reflexes were absent. The arm muscles were all flabby. There was marked weakness

of all the muscles of the arm. The patient's grip was excellent. All reflexes were hyperactive. The leg muscles were all flabby. No sensory changes could be made out.

The eye findings and history, obtained on May 8, 1929, were: The eyes tired after a half hour of near use; the print blurred. No headaches occurred. Distant vision was slightly blurred with glasses. Vision was very changeable. The right upper lid had drooped at times for eight months. Usually this condition was absent in the morning. The patient saw double about Christmas, 1928; at that time he had a vertical diplopia which later became horizontal. There was no family history of a similar condition.

The left palpebral fissure was larger than the right. The left upper lid cut off a 1 mm. arc of the cornea from 11 to 1 o'clock. Voluntarily the patient could raise both upper lids so that the sclera showed above the corneas, slightly more above the left cornea. From 3 to 4 mm. of the right sclera was still visible on extreme right abduction. There were a few occasional nystagmoid jerks when the patient looked to the right. The pupils and accommodation were normal. Con-

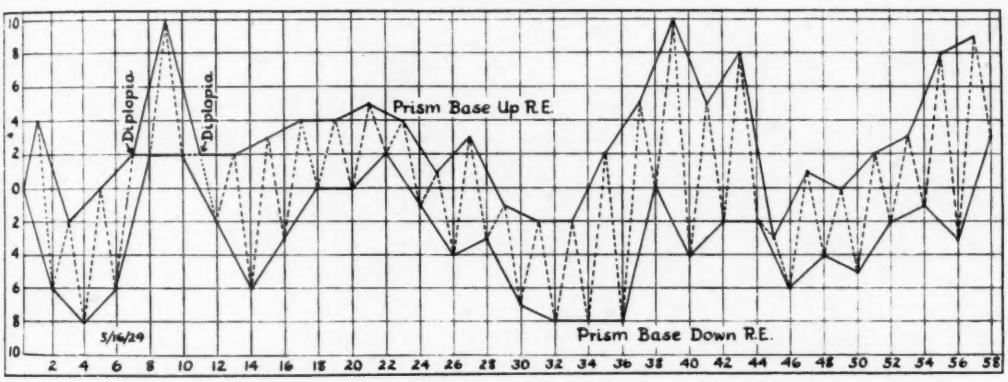

Chart 3.—Note the marked irregularity in the readings, alternating between groups of opposite character (compare with chart 1).

vergence was normal. The cover test was negative for divergence or convergence. Grossly the fields were normal. Only questionable temporal pallor of each disk was noted.

Muscle tests showed from 7 to 10 prism diopters of esophoria. Hyperphoria was variable; sometimes right and sometimes left hyperphoria was found, changing from moment to moment, though left hyperphoria was present most often. The amount varied from 0 to 4 or even to 6 diopters of right or left hyperphoria. The findings were similar whether the Maddox rod was before the right or the left eye. No cyclophoria was present. Abduction (prism base in) amounted to 8 prism diopters; adduction (prism base out) to 24 prism diopters; right and left sursumductions varied from 2 to 12 prism diopters. It was impossible to plot any muscle defects by the Hess chart.

Homatropine refraction showed: in the right eye, with a +2.25 diopters sphere +0.50 cylinder axis 75, vision of 0.8+2; in the left eye, with a +1.25 diopters sphere +0.50 cylinder axis 75, vision of 0.8+3. The fundi, media and disks were normal. A postcycloplegic examination on May 16, with full correction showed: in the right eye, vision of 0.4+2; with 0.50 diopter less, 0.6+2; in the left eye, vision of 0.3; with 1.00 diopter less, 0.6. With full homatropine correction

the patient had a near point of 7.5 inches where he could read the smallest Jaeger test type for fifteen minutes without tiring or without the print blurring. (The results of muscle tests on this date are given in chart 3.)

On May 8, 1929, the date of the first examination by the special muscle test, I made the following notes: "Muscle test shows an unusual condition. I believe that the eye muscles are not at all weak, but that impulses are at the base of the eye trouble, possibly the general condition." On May 16, the following note was made: "The eye under postcycloplegic examination acts just like many other cases with spasm of the accommodation, in that distant vision is temporarily blurred, during which period less plus is accepted."

On June 19, 1930, muscle tests showed that single binocular vision was present at the onset of the test. By the Maddox rod test the patient showed 20 prism

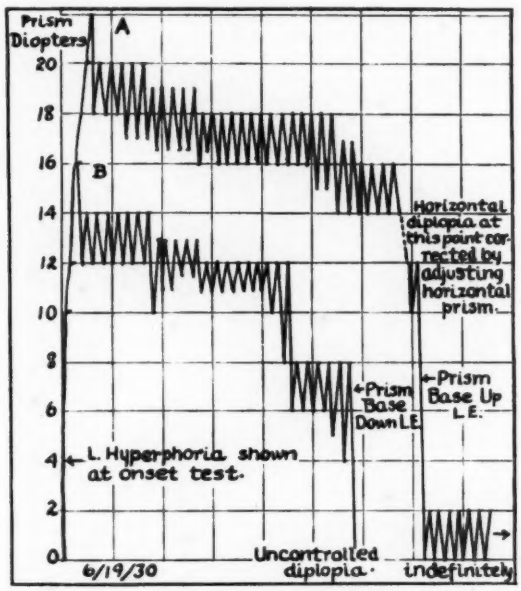

Chart 4 (case 2).—This shows sursumductions as taken separately; that is, curve *A* is obtained by first taking the right sursumduction, then reducing the prism only sufficiently to restore single binocular vision and then increasing again to diplopia. After a rest, the left sursumduction (*B*) is similarly taken. Note the similarity in the two curves and the small amount of reduction in prism necessary to restore single binocular vision. Compare this small amount of prism with the large amount initially required to produce more than momentary diplopia. This finding is in agreement with the small separation of the images and the amount of hyperphoria which varies from 4 prism diopters of left hyperphoria to 23 prism diopters of right hyperphoria in the few times taken (see also chart 8 *C*).

diopters of right hyperphoria on the first reading. On repeating the test immediately the reading was 4 prism diopters of right hyperphoria. No rest was given and the ductions were taken immediately (chart 4). Horizontal diplopia developed and a rest period of three minutes was given. Following this, without any manipulations, spontaneous diplopia occurred after one-half minute of simple observation with the eyes open and fixed at 20 feet. This diplopia was corrected by 6 prism

diopters, base out, and 6 prism diopters, base up, before the left eye. On repeating the Maddox rod test at this time, 6 prism diopters of esophoria and 23 prism diopters of right hyperphoria were found. The results of these and similar tests of the heterophorias and the ductions are better shown in chart 4.

The observer was more tired than the patient at the end of seventy minutes of testing. The palpebral fissures which were usually narrower than normal, and were so before the tests, were exceedingly wide; from 2 to 3 mm. of sclera showed above the limbus of each eye, and both eyes had a staring expression. With two minutes of rest the palpebral fissures were back to normal (left) and less than normal (right). By having the patient look up and down about twenty times with both eyes open, the expected (and previously obtained) ptosis did not occur; instead an actual lid lag was produced with definite widening of the palpebral fissures. The question is raised whether or not this reversal of the ptosis may be interpreted as an indication of an improvement. (The patient definitely improved clinically while on suprarenal extract.)

CASE 3.—W. M., the patient, a woman, aged 38, gave the following history on March 13, 1930. Present Illness: The patient complained of diplopia for two years and weakness and headaches for one and a half years. In August, 1928, she became conscious of a visual defect; she went to an oculist and had glasses fitted. In a few weeks she saw double, especially when nervous or fatigued. Some of this diplopia continued all day. Six months later the upper extremities became weak. At first the patient dropped things. The fatigue was more noticeable when she tried to play the piano or comb her hair. Early in 1929, before the weakness was noted, the patient found that her eyelids drooped; they felt heavy and tired. The drooping was observed more often when she was under some nervous strain and rarely when she was resting or alone. On Jan. 30, 1929, the diagnosis of myasthenia gravis was made. Except for the history of fatigue and weakness, no general weakness was found; in fact, the muscles were well developed and showed good power. There was nothing important in the history in relation to the nervous system except flushing and nervousness.

General physical examination showed nothing of any importance, except possibly a palpable thyroid, flushing of the skin, active reflexes and a variable unsustained ankle clonus. The eye examination showed a bilateral ptosis. Opening and closing the lids fifteen times definitely fatigued them. The pupils were round and equal and reacted normally to light and in accommodation. External ocular movements were full. Nystagmoid movements were present when the eyes were held to the right or left. The fundi were normal. A further history revealed that the original symptom was vertical diplopia, intermittent in character and lasting two months. This diplopia was less frequent after the patient obtained glasses. When she was first seen by me, and again the next day, no ptosis was noted, but this could easily be produced by opening and closing the eyes ten times. For intervals of several moments the patient could scarcely open the eyes. The candle test showed a variable defect, fairly consistent for paresis of the right abducens (?). The nystagmoid movements previously noted were inconstant. The results of muscle tests at 20 feet are shown in chart 5. Tests conducted at 13 inches indicated unusually strong duction power. Abduction was 48 prism diopters and adduction 36 prism diopters; right and left sursumductions were 24 prism diopters.

The instability of the muscular condition is indicated by the various findings of the ophthalmologist who had previously examined the patient. His observations in part were: on Feb. 15, 1929, left hyperphoria; on March 6, right hyperphoria, and on March 29, left hyperphoria. Similarly irregular findings are reported in

Posey's case 1 in which at one time 7 prism diopters of right hyperphoria was found (in November, 1918), and later 3 prism diopters of left hyperphoria (in April, 1919), while several weeks later orthophoria was present. One neurologist, after examining this patient said, "I will agree to a diagnosis of myasthenia when that means an easily fatigued individual, but as myasthenia gravis is a definitely defined clinical picture with symptoms this patient does not present, I do not believe this should be diagnosed myasthenia gravis." Another neurologist said: " . . . on Jan. 30, 1929 . . . I made a diagnosis of myasthenia gravis. There was a partial ptosis and a tendency to diplopia. . . . I saw the patient last on Feb. 18, 1930, when I found her considerably better. However, when she looked upward for half a minute the left lid drooped and diplopia set in."

When I examined the patient, I made the following notes: ". . . unusual condition practically identical with but two other cases examined with these

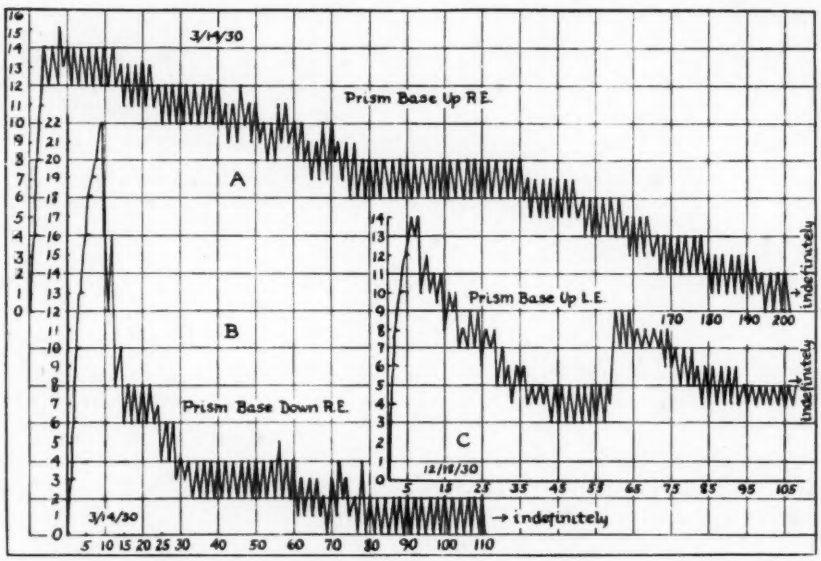

Chart 5.—These are high duction readings in the vertical meridians both up and down (right and left sursumductions). A correction for a horizontal defect was made while readings were obtained in the vertical meridian. Note that the patients see singly with as much as six sevenths of the maximum amount of prism required to produce diplopia. *A*, *B*, tests in case 3; *C*, tests in case 4.

instruments (phorometer and Maddox rod) out of over 500 tests. Both of these cases were seen one year ago and were typical cases of myasthenia gravis. My impression then was, as it still must be, that whatever myasthenia gravis is, there is no real myasthenia of the eye muscles, but rather exceptional increased stimuli reach the muscles, varying momentarily. I consider a vasomotor disturbance or a neuromuscular change on a vascular basis, possibly central, as the basic pathology."

When last heard from, in May, 1931, the patient had gained weight and was feeling fine. She continued to take suprarenal extract, which she had been taking for eight or nine months. She could drive her car. When tired or excited, she still had diplopia.

CASE 4.—V. L., the patient, a man, aged 42, was first seen on Sept. 23, 1930. Present Illness: The patient complained of diplopia for distant and near vision for over ten years. He sometimes had no diplopia for periods of four or five months, but then had a recurrence on emotional or nervous disturbance. He usually walked with one eye closed to avoid diplopia. Early in the morning he had no diplopia. Three or four years before, the left upper lid drooped for three or four days, but there had been no trouble since. He saw an oculist five or six years before, and glasses were prescribed, which were worn only one week. The images were usually above one another; the image seen by the left eye seemed higher. The patient came from a family of ten of whom one was a half-sister. Two brothers died shortly after birth, another of an accident and another of insanity eight years following sunstroke (at the age of 28). All the others were living and well. The patient felt that he was in perfect health, except for the diplopia. No other muscles had been weak, although the patient was conscious at times of some peculiarity of facial expression, some disability in smiling. Medical examination gave entirely negative results.

An eye examination on Sept. 13, 1930, showed: In the right eye vision was 1.5+1; in the left eye, 1.5+2; slight alternating divergence was present at 14 inches. The pupils were slightly derounded, but reacted satisfactorily. The tactile tension and disks were normal. On September 24, refraction under homatropine showed: in the right eye, with a +0.50 cylinder axis 90, vision of 1.5+1; in the left eye, with a +0.25 diopter sphere +0.25 cylinder axis 60, vision of 1.5+1. The pupils were equal and round. The right disk was normal; the left disk showed a narrow rim of circumpapillary atrophy. The fundi and media were normal. The Hess test chart only suggested a right inferior rectus paresis. The candle test, too, was suggestive of this. On October 1, the Maddox rod test showed that the right eye required 5 prism diopters, base in, and 12 prism diopters, base down (exphoria and right hyperphoria).

On December 18, duction tests were done for the first time. The patient showed 9 prism diopters of right hyperphoria and 6 prism diopters of exophoria. Abduction was 12 prism diopters; adduction, 14 prism diopters; right sursumduction, from 1½ to 8 prism diopters; left sursumduction, from 2 to 4 prism diopters. On repeating the Maddox rod test a right hyperphoria of 5 prism diopters was found. The candle test still suggested a right inferior rectus paresis. The pupil reactions and accommodation were normal. The tests are shown in detail in chart 5*C*. After a few hours rest the patient was reexamined and the following notes were made: The palpebral fissures were not materially changed by exercise. The width of the palpebral fissure with the eyes in the primary position was 8.5 mm. (right) and 8.0 mm. (left); with fixation upward, 11.0 mm. (right and left); with fixation downward, 4.5 mm. (right) and from 4.0 to 4.5 mm. (left). "Crowfeet" were present. The orbicularis showed a peculiar condition in the lower lid of each eye. There seemed to be a negative contraction of this muscle, pulling the lower punctum temporally and down, with a "knotting" of the muscle below the external canthus on each side. On smoothing the skin and removing the finger, one could see the "wormlike" retraction of the muscle. When the patient began to close the eyes, one could see this muscle straighten out and each punctum move toward the eyeball, an action analogous to that of a cat preparing to spring. On closing the eyes, the patient showed much visible twitching of the lids which he was unable to control. There was no tendency to spontaneous diplopia at this time in three or four minutes. No sign of ptosis was present (the upper lids were very short). The lids did not retract visibly on

attempting to widen the palpebral fissure while the patient was looking straight ahead, even though the frontalis muscles contracted. There was a deeper groove on the left than on the right side of the forehead (root of the nose) on frowning. When the patient smiled, there was also a slightly shallower wrinkle on the right than on the left side, and the left side of the lip was definitely higher than the right. The corneal reflex was definitely decreased in each eye. A few hours previous to this examination there was only a mild nystagmus present in each eye on looking to the left. Now an irregular swaying vertical nystagmus could be elicited on looking up. The right eye seemed to stop rotating to the left while the left eye continued. Though the patient stated that he had done much work with the sledge hammer years before and was right-handed, there was a slight increase in the strength of the left hand over that of the right. "The muscle findings in this case are practically identical with those I have noted in all three definite cases of myasthenia gravis thus far tested *and in no other condition.* The history is suggestive of a mild myasthenia gravis with remissions, with chiefly an involvement of the eye muscles. Referred to neurology department for study and opinion."

The same neurologist who questioned the diagnosis of myasthenia gravis in case 3 on Feb. 6, 1931, said, "Believe this may be included in the syndrome of myasthenia gravis. Advise increasing drop doses of nux vomica." Because the basal metabolic rate was —15 per cent, it was thought desirable to see how the patient reacted to thyroid medication. This was tried with and without compound solution of iodine, but the diplopia grew definitely worse and medication was stopped, with improvement of the eye condition. The patient was placed on strychnine sulphate, one-thirtieth grain (2.1 mg.) three times a day, for several weeks, with no change in his condition. Because of a long remission while potassium iodide was given several years before, this drug was added to the strychnine, and improvement was reported by the patient.

CASE 5.—R. R., the patient, a woman, aged 24, gave the following history on March 24, 1931. Present Complaint: The patient suffered from double vision, weakness in the arms and legs and dizziness. The first symptom to attract attention was diplopia, which had been present for three years. Weakness in the arms and legs had been present for over a year. There had been no difficulty in eating, drinking or talking. No asymmetry, atrophy, tremors, ataxia or pathologic reflexes were found. The abdominal reflexes were present. No sensory disturbances were noted.

Eye Findings: The pupils were round and equal, and reacted normally to light and in accommodation. Nystagmus was present, especially marked in the right eye and on looking to the right. The upper lids drooped. There was no ocular movement upward.

An examination on April 1, 1931, showed corrected vision of 0.6+2 in the right eye and 1.2+3 in the left. The left palpebral fissure was slightly wider than the right. The left eyeball (in the primary position) was about 2 mm. higher than the right. On prolonged fixation both upper lids drooped. There was no divergence apparent or under cover. Extra-ocular movements of both eyes were definitely limited upward. The patient was able to elevate the eyes a small distance above the horizontal plane, the left eye remaining higher than the right. There was some limitation of abduction as well as adduction, more marked on the left than on the right. There were coarse horizontal nystagmoid movements, especially on looking to the right. The fundi and media were normal. The results of muscle tests were variable (examination at noon). The Maddox rod test initially

showed 2 prism diopters of left hyperphoria and 14 prism diopters of esophoria. Ductions were difficult to take owing to varying horizontal diplopia. Left sursumduction was from 4 to 8 to 12 prism diopters and right sursumduction from minus 4 to 0 to plus 3 to plus 5. The test was incomplete, but suggested findings similar to those in other cases of myasthenia gravis (chart 8 *B*).

CASE 6.—P. M., the patient, a man, aged 21, was seen in the eye clinic on Feb. 14, 1931. History: In 1923 or 1924, he had drooping of the eyelids for about one month, but no diplopia. Two years later a similar attack of ptosis occurred. In 1928 there was another period of ptosis for three or four months, with diplopia on looking straight ahead; the patient held his head tilted to the right and back to overcome this. Prisms were prescribed. There was no nystagmus, and no history of related illness or injury was obtained. Ptosis had been rare since 1928 and had occurred only when the patient was tired. In October, 1930, in addition to diplopia at distance, he began to have trouble in swallowing and talking; this had been more marked since January, 1931.

Eye Findings: The palpebral fissures were equal (9.5 mm. under electric illumination with the eyes in the primary position; 8 mm. in daylight). The pupils

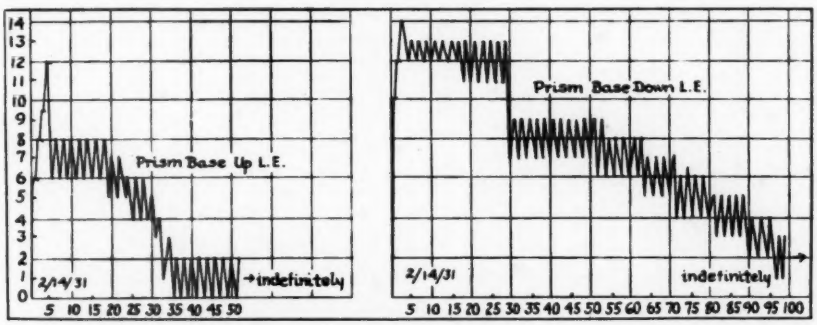

Chart 6.—Vertical phorias vary during the test between 6 prism diopters of right hyperphoria and 6 prism diopters of left hyperphoria, and in character are dependent on the kind of stimulation. Note that the patient sees singly with from two thirds to three fourths of the prism required to produce diplopia (after the production of the diplopia).

measured 4 mm.; they were equal and round and reacted normally to light and in accommodation. Exophthalmometer readings were: right, 18 mm.; left, 17 mm. When the patient closed the eyes as in sleep there was a 1.5 mm. area of sclera still visible in the left eye and about 3 mm. in the right eye. On squeezing, a definite weakening of the orbicularis was evident. There seemed to be marked impairment of the elevators of both eyes. Otherwise the ocular movements were essentially normal except for nystagmoid movements on looking to the right, left and up. The quick component was in the direction of the gaze. The fundi and media were normal. Vision was 1.5+ 4 in the right eye and 1.5+ 2 in the left.

Muscle tests on Feb. 14, 1931, showed initially no hyperphoria and 6 prism diopters of esophoria. Abduction was 3 prism diopters, adduction 38 prism diopters, left sursumduction 1 prism diopter and right sursumduction 8 prism diopters. Further testing gave left sursumduction 4 and right sursumduction 0. The Maddox rod test showed 2½ prism diopters of left hyperphoria. On February 17, tests showed 3½ prism diopters of left hyperphoria and 4½ prism diopters of esophoria; abduction was 5 and adduction 22. Sursumductions are shown in chart 6. Candle

tests indicated a right and left abducens paresis but the tests were inconstant. On February 19, the record stated: "I note a decreased ability to smile, and the upper lip scarcely moves on talking. Speech is slurred and indistinct. On suprarenal extract for six weeks the patient believes he made a marked recovery, but since stopping this medicine he has been definitely worse. He has a definite staring appearance, and lid lag is present. The patient has no trouble with accommodation. His near point is 4-5 inches."

Case 7.—In M. J., the patient, a woman, aged 23, the onset occurred in 1928 (at the age of 20) with weakness in the hands, difficulty in chewing and swallowing, diplopia and ptosis. The first impression was that status postencephaliticus was present, but the diagnosis was changed to myasthenia gravis. The patient went to an ophthalmologist early on account of diplopia. Eye Findings: The right palpebral fissure measured 10 mm.; the left, 8 mm. There was some difficulty in looking up and a definite intermittent impairment of motility. There was no spontaneous diplopia for distance. The right pupil was slightly larger than the left; both reacted normally to light and in accommodation. The fundi and media were normal. The accommodation and near point were normal.

Muscle Tests: The Maddox rod test showed 1.5 prism diopters of left hyperphoria and 2 prism diopters of esophoria. Abduction was 6 prism diopters, adduc-

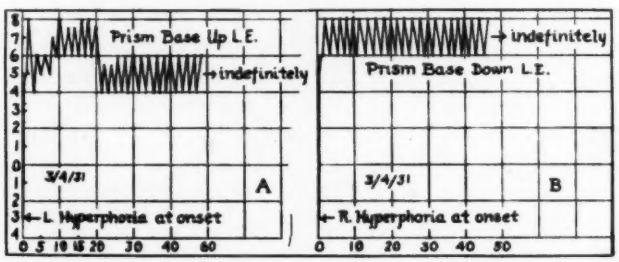

Chart 7.—*A*, at the onset the Maddox rod test shows 3 prism diopters of left hyperphoria. At the first maximum reading (base up, left eye) this test shows 2 prism diopters of right hyperphoria. At the end of the curve this Maddox rod test shows 3 prism diopters of right hyperphoria. *B*, at the onset the patient shows 3 prism diopters of right hyperphoria. At the first maximum reading (prism base down, left eye) the test shows 3 prism diopters of left hyperphoria. At the end of the curve the Maddox test shows 5½ prism diopters of left hyperphoria.

tion 14 prism diopters, right sursumduction 0 and left sursumduction 4 prism diopters. Repetition of the Maddox rod test revealed 2 prism diopters of left hyperphoria. Right sursumduction readings were 3, 6 and 7 prism diopters; the Maddox rod test now showed 2 prism diopters of right hyperphoria. Left sursumduction readings were 4, 5 and 7 prism diopters, and the Maddox rod test showed 3 prism diopters of left hyperphoria. Further readings are shown in chart 7.

The record stated: "Muscles tested with the faradic current give a myasthenic reaction to stimulation; the muscles contract for a time, then relax suddenly and after a short interval contract again, the current running continuously."

Case 8.—J. H., the patient, a colored woman, aged 19, seen in January, 1930, had an advanced case of myasthenia gravis; ptosis had occurred at the onset in 1928. Phoria and duction tests were not possible because of the patient's bedridden condition. On Jan. 6, 1930, the external ocular movements were recorded as normal. Ten days later, a slight oculomotor nerve paresis and an abducens paresis

were noted. The conjugate upward movement was absent. There was only a slight fatiguing on stimulation with the faradic current (negative reaction of Jolly). This case is added because of the definite finding of what seems to be a weakness of the accommodation. The successive readings for the near point with both eyes open were 9, 11, 13, 15 and 17 inches; the patient refused to continue. Whether these findings were due to poor cooperation and marked general exhaustion or to a definite weakness of the accommodation is not clear.

SUMMARY OF CASES

Syphilis and tuberculosis were ruled out as completely as possible by the usual laboratory tests. No serologic tests were made in case 5 because the patient failed to return. Unless otherwise stated, the vision in each case was normal. The fields and tension were also normal. Roentgenograms of the skull and chest revealed nothing unusual. The cases fall definitely into the group of myasthenia gravis, all but two of the progressive type (cases 3 and 4). Cases 3, 4 and 6 seem to remain bulbar in character (case 4 for over ten years). In five of the eight cases diplopia was the first definitely noted symptom, though in case 1 vague fatigue symptoms and temporary loss of the voice occurred several years previously and in case 7 the diplopia was not the only initial symptom. In every case diplopia was present when the definite picture of myasthenia gravis developed. Ptosis was definitely recorded as being present at the onset, with diplopia in one case (case 3); it preceded the diplopia and other symptoms by several years in another case (case 6) and was present at the onset with diplopia and other symptoms in case 7. In all other cases ptosis was present at some period of the disease.

In a consideration of the pathologic basis for this condition, one is impressed by a rather unique situation in that here is a well defined clinical entity in which cases with marked symptoms coming to autopsy show few positive indications. The main, though not consistent, observation is that of a round cell infiltration around the blood vessels, chiefly in muscles but also in the central nervous system, the endocrine glands and elsewhere. These areas were called lymphorrhages by Buzzard. Muscular atrophy, the expected finding, has been noted only in the extremely advanced chronic cases, in which disuse has been considered the more likely cause of the atrophy. Occasional changes in the central nervous system (McAlpine [13]), such as mucoid degeneration, cortical atrophy and chromatolysis of the ganglion cells, particularly those of the medulla, do not seem sufficient to explain the phenomena.

Studies on the electrical responses have been carried on for a long time. In 1894, Jolly found that the muscles of a myasthenic person

13. McAlpine, D.: A Form of Myasthenia Gravis with Changes in the Nervous System, Brain **52**:6, 1930.

quickly failed to respond to stimulation of the motor nerves by the faradic current, while the response to the galvanic current continued to be normal. This "reaction of Jolly" has been considered of diagnostic importance. However, it is not always present in these cases (Campbell and Bramwell, Keschner and Strauss and Dana) and, as Posey pointed out, it is present in conditions other than these, such as muscle hypertrophy, chronic poliomyelitis and Landry's paralysis. Marinesco, Sager and Kreindler referred to this reaction in normal persons after stasis following the application of an Esmarch bandage. However, Wolff, Keutman and Cobb [14] said that records of muscles with venous stasis are not sufficiently varied from the normal to permit such views. The work in this field for the most part has been undertaken with the

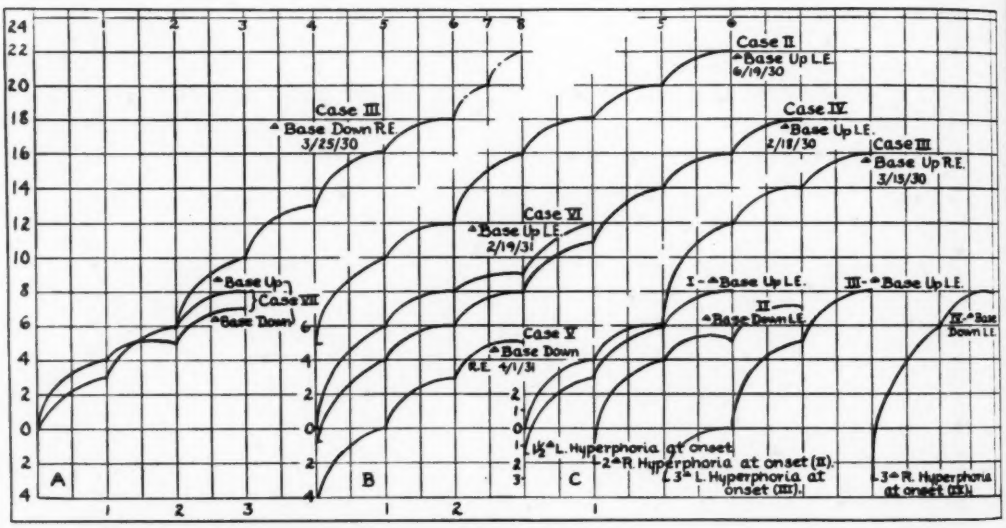

Chart 8.—Summation effects obtained in cases 2 to 7. Note that the increments are relatively equal in amount to the normal contraction (from 2 to 4 prism diopters). The end of each "contraction" is indicated by a momentary diplopia, which is characterized by (1) the promptness with which it is overcome, and (2) the small separation of the images (from 1 to 3 cm. at 20 feet). The final readings are large in both directions; viz., base up and base down before the same eye (shown, for brevity, only in cases 3 and 7, though present in the other cases). In *C* are several consecutive readings with the amount of hyperphoria present before each step. Note the relation of the "hyperphoria" found to the previous duction. The last (IV) reading taken is that of the left sursumduction. At its onset 3 prism diopters of right hyperphoria is present; at its conclusion 5½ diopters of left hyperphoria is present.

a priori assumption of a "weak" muscle, and the results have been interpreted with this in mind. The fatigue of the muscles in myasthenia

14. Wolff, H. G.; Keutman, H., and Cobb, S.: The Electromyogram in Myasthenia Gravis, Brain **51**:508, 1928.

gravis is different than in the normal (Wolff, Keutman and Cobb, Cobb and Forbes,[15] Herzog and others). Marinesco, Sager and Kreindler presented plethysmograms of myasthenic muscles and interpreted the results as indicating a vasodilatation, failing to consider the possibility of the results obtained being due, even in part, to a change in the contraction state of the muscle itself. Among the numerous theories as to the cause of the underlying pathologic changes in myasthenia gravis may be mentioned those considering the condition to be due to abiotrophy, toxin, heredity, infection, endocrine dysfunction or a disturbance of the vegetative nervous system. Marinesco, Sager and Kreindler gave a comprehensive discussion of the arguments in favor of the last-mentioned theory. They claimed that ergotamine aggravates the myasthenic symptoms. Klar referred to experiments by von Boehm with proveratrin and by Hoffman with curare in which the reaction of Jolly was obtained. The myautonomy found by Rautenberg,[16] too, does not necessarily speak for a muscle weakness but rather for a loss of orderliness between impulses and response. In a consideration of whether the condition is of myogenic or neurogenic origin, Herzog gives two possibilities: (1) that the normal rhythmic impulses to the muscle are weaker and weaker, so that the reaction current from the muscular contraction is weaker, and (2) that the innervation is normal but the muscle responds less and less to the normal impulses. Herzog favored the latter and did not mention a third possibility, that all the muscle fibers may not be stimulated by every impulse and the response may be less and less as fewer fibers are left to respond, the previously stimulated fibers failing to respond, *due possibly to a protracted contraction state*. That the muscle is not at fault seems probable from the finding that myasthenic muscles respond to: (1) a change in polarity in faradic stimulation, (2) continued stimulation with a faradic current after a brief "refractive state," (3) a change to a galvanic current, and (4) voluntary effort after failure to respond to a faradic current. If one assumes that the muscle is not at fault in myasthenia gravis, and that the stimulus to contract reaches the muscle, one must assume, then, that the muscle fails to contract because it cannot contract, and, if this is true, that it cannot contract further because it has already contracted maximally or because the influence antagonistic to the contracting stimulus is equally great.

Continuing in the realm of theory, one may say that if one accepts the dual innervation of muscle as established, it could well be that the contracting stimulus via the spinal or cranial motor nerves reaches the

15. Cobb, S., and Forbes, A.: Electromyographic Studies of Muscular Fatigue in Man, Am. J. Physiol. **65**:234, 1923.

16. Rautenberg, E.: The Pathologic Physiology of Human Skeletal Muscle, Deutsches Arch. f. klin. Med. **93**:389, 1910.

muscle normally and the muscle contracts as it should (the contractile part of the sarcoplasm is normal), but that the necessary stimulus to relaxation (via the sympathetic [?]) is not normal or does not reach the muscle (the reflex inhibitory pathways are not functioning normally). If this view is correct, it would fit in with the well supported view that myasthenia gravis is due to pathologic changes primary in the vegetative nervous system.

In charts 1 to 8 the data obtained in cases of myasthenia gravis are compared with similar data obtained in cases with and without heterophorias. Among about 1,000 such tests done there were at least six cases of epilepsy, seventeen cases of chorea, six cases of paralysis agitans, 6 cases of chronic encephalitis, one case of encephalitis with oculogyric crises, one case of epilepsy after subacute encephalitis, nineteen cases of multiple sclerosis, three cases of progressive muscular atrophy, one case of progressive muscular dystrophy with unusual sensory involvement, one case of pernicious anemia with cord changes and about twenty cases of syphilis of the central nervous system.

Only in myasthenia were the results markedly variable as to the demonstrable heterophoria or the duction. Although in the light of Sherrington's principle of reciprocal innervation, relaxation of antagonists is an important element in the contraction of a muscle, the major work, in maintaining fusion, for example, is done by the contracting muscles. This and the "modus operandi" of prisms force the interpretation of the data given in charts 1 to 8, as indicative of abnormally strong muscles with astonishingly increased and continued activity. The large reading in charts 2, 3, 4, 5 and 6 support the possibility of a cumulative contraction of the stimulated muscle or muscle group. This idea is supported by the increments given in chart 8 as compared with the normal readings and by the findings with the Maddox rod, which indicate that a state of abnormal contraction of the stimulated muscle existed even with the fusion faculty suspended; a newly created "hyperphoria" is found that is of an amount in keeping with the amount of stimulation (chart 8 *C*). This is further supported by the amount of separation existing when diplopia is produced. This separation in myasthenia may be as little as 1 cm., even though the diplopia is produced by as much as 20 prism diopters. In normal subjects, when diplopia is produced by only 2 prism diopters, the separation of the images at 20 feet is more than 10 cm., and any increase in the prism power increases the separation proportionately. The data here presented, so far as I am aware, are the first clinical presentation of the summation phenomenon in muscles (chart 8). That the muscles presumably contracting are actually doing so is demonstrated by case 3, in which the eye behind the prism was seen to rotate, and this rotation was in the direction of the apex of the prism, increasing and decreasing with an increase and decrease in

the amount of prism. This clinical presentation of the summation phenomenon is based on the data obtained by the use of prisms to change the light rays. The responses were truly superimposed responses on the average maximum, the difference being that in the normal person further stimulation yielded no such responses as those obtained in myasthenia gravis. The maximum "single" contraction on stimulation with prisms is comparable to that obtained on electrical stimulation; in neither case does one obtain a contraction involving the entire capacity of the muscle.

CONCLUSIONS

1. The irregularity in the amount and quality of the heterophoria in the short span of a few moments, the irregularity in duction readings, the unusually high duction readings, the close proximity of the images when diplopia is produced, the approximation of the amount of prism used in creating the diplopia and the hyperphoria as measured by the Maddox rod test and the actual turning of the eye in the direction of the apex of the prism, indicating that the muscle stimulated does not "tire," permit of but one interpretation, namely, that the muscle or muscles stimulated contracted, but failed to relax promptly or completely, remaining in a prolonged contraction state.

2. Such findings obtain in no other condition in which tests have been performed, and therefore would seem of diagnostic value in this condition.

3. From these findings I am led to conclude that myasthenia gravis is not a true myasthenia.

SIZE AND SHAPE OF OCULAR IMAGES

II. CLINICAL SIGNIFICANCE

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AND

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INTRODUCTION

The number of patients showing ocular symptoms and the many showing general symptoms of possible ocular origin that have received no relief from refractive treatment, nor even from some admirable mental therapy, form quite a group in the practice of every ophthalmologist. In a certain percentage of these cases a diagnosis of ocular neurosis can easily and correctly be made, but in many cases such a diagnosis does not meet the situation. This haven is often a greater source of contentment to the physician than to the patient. Ocular neurosis is an important factor, but one seems prone to place too large a number of cases in this convenient pigeonhole.

Two years before this writing our interest was aroused in the work of Professor Ames and his associates, and since that time we have endeavored to learn to what extent his investigations fit into the clinical picture. This interest was brought about because of our inability and also the inability of abler men than we to give relief in certain types of cases. In general, these cases include those with or without refractive error which do not seem to fall in the neurosis class, cases of photophobia without apparent physiologic cause and cases of muscular imbalances that do not respond to treatment either by prisms or operation. Most oculists recognize that such treatment does not give permanent relief in all cases. Practically all cases in this report were referred cases; examinations had been made and refractions performed by one or more reputable ophthalmologists. In all of them ocular treatment, and in many cases mental treatment, had been given without result. For this reason, the possible existence of some physiologic factor, heretofore overlooked, seemed to justify a careful investigation to account for some of these disturbing symptoms. Surely, a

Submitted for publication, Dec. 3, 1931.

From the Department of Research in Physiological Optics, Dartmouth Medical School.

Read before the American Academy of Ophthalmology and Otolaryngology, French Lick, Ind., Sept. 17, 1931.

diagnosis of neurosis should not be made until it is determined that there is no physiologic cause for the disturbances.

We have been particularly fortunate in our ability to follow up the cases in which measurements were made. This may be partially attributed to the fact that no limit has been put on the amount of care and time taken in the examination in these cases; furthermore, up to the present no charge has been made for such examinations.

With this brief introduction, we shall go on to a description of the method of examination.

METHOD OF EXAMINATION AND PRESCRIPTIONS GIVEN

The patients measured were, in general, those who had not been relieved by previous refractions. Some had had a number of refractions made by different ophthalmologists. Their discomfort had persisted. Disease of the eye had been ruled out, and various other possible causes, not ocular, had been eliminated. One group of patients did not show strictly ocular symptoms. Some showed a record of headaches of obscure origin which both internist and ophthalmologist had investigated; others had stomach disorders of apparent nervous origin; others had various general nerve reflex disturbances. This entire group had been examined for ocular defects, and that factor as the chief or contributing cause had been considered. A second group of patients had symptoms that were manifestly ocular: photophobia, marked fatigue, bulbar aches and pains, and headache in the ocular regions. A third group included those patients who had a combination of ocular and nonocular symptoms, who showed signs of stress and discomfort somewhere along the sympathetic tract.

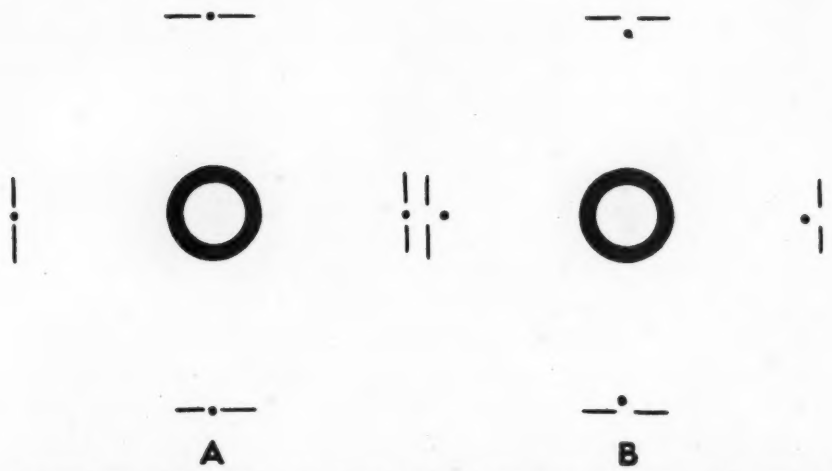
The first part of the routine is that generally followed in regular practice: the taking of the history, ophthalmoscopic examination and objective and subjective tests for refraction and muscle balance. With this information measurements on the instrument are begun. The design and the principles underlying the operation of the instrument have been described in a previous paper.[1] It is adjusted quite accurately and set for the patient's interpupillary distance. Care is taken to place the patient's head in the correct position. With these preliminary but quite important details attended to, the measurements proper begin. They are taken at 20 feet (0.164 diopter) and at 16 inches (2.50 diopters).

The patient's refractive condition is measured first; that is, with binocular fixation at 20 feet, the point in space that is conjugate to each retina is determined.

1. Ames, A., Jr.; Gliddon, G. H., and Ogle, K. N.: Size and Shape of Ocular Images: I. Methods of Determination and Physiologic Significance, Arch. Ophth. **7**:576 (April) 1932.

Then determination is made of the size of the ocular image in one eye relative to that in the other eye at a distance of 20 feet. The method of determining size differences was described in a previous paper.[1] If the ocular images in both eyes are the same size, the lights will fall in the breaks of the lines (see chart *A*). If the images are not equal, the lights will be displaced relative to the lines. Thus, if the top light is below the top line and the bottom light is above the bottom line, the left eye image, which is represented by the distance from line to line, is larger vertically than the right eye image, which is represented by the distance from the top light to the bottom light (see chart *B*).

In this way it is determined whether the images are equal in size, or whether one image is larger overall than the other or only larger



Appearance of targets. (*A*) when ocular images are the same size, and (*B*) when image of left eye is larger than image of right eye.

in one meridian than in the corresponding meridian of the other eye. Various combinations of these conditions are possible.[2]

The next step is to correct any errors that have been found in the patient's refraction and size of image. The errors of refraction are corrected by lenses from the trial set. Any difference in size is equalized by the "size" lenses described in a previous paper.[1] However, these errors are not corrected independently of each other.

2. The position of the lights relative to the lines also determines the nature of the patient's slip phoria or fixation disparity.[3] In exophoric slip the lights are shifted to the left relative to the lines; in esophoric slip the lights are shifted to the right.

3. Ames, A., Jr., and Gliddon, G. H.: Ocular Measurements, Tr. Sect. Ophth., A. M. A., 1928, p. 1. Ames, A., Jr.; Ogle, K. N., and Gliddon, G. H.: Corresponding Retinal Points, the Horopter and Size and Shape of Ocular Images, to be published.

Refraction and size must be corrected at the same time by the combined effect of the two types of lenses. For instance, a change in the refractive lens before either eye usually affects the size of the ocular image. If a + 0.25 diopter sphere is added to the right eye to bring about equality of refraction, the magnification due to the plus power in the lens increases the size of the image in that eye by about 0.50 per cent. A redetermination of size has to be made and the size correction modified. When the refraction is balanced and the size of the ocular images equalized, the combined refractive and size lenses before the eyes are considered to be the correction necessary for that particular distance.

When correction at 20 feet has been made, the procedure is repeated at 16 inches (2.50 diopters). If the patient is presbyopic, his reading addition is given. If the correction found for near differs from that found for distance, the patient is rechecked for distance. When satisfactory correction has been found for distance and for near, temporary glasses are made up and given to the patient to wear overnight, and he is asked to use his eyes as much as possible. The tests are repeated the next morning. It is usual to find that some adjustment has taken place and that some refining of the correction is necessary.

The final prescription is a combination of refractive power and size correction. This may be put in one lens if the size increase needed is spherical (or overall) and less than 1.75 per cent. Such an increase is obtained by grinding one lens on a base curve that is greater than that of the other. A lens on a 12.00 diopter base curve magnifies the size of the image 1.60 per cent as compared with a flat lens. Various degrees of spherical increase up to 1.60 per cent may be had by the proper choice of the base curves for the two lenses. For a spherical increase above 1.75 per cent and for an increase in one meridian only, it is necessary, at the present time, to use an auxiliary lens. This is put into a rim and attached to the frame in such a way that it may be removed for cleaning. It is hoped that within a short time all size corrections may be combined with refractive prescriptions in the one lens. The first attempt has been made and a few lenses of this type are now being worn.

In connection with the procedure mentioned, the following observations are of interest.

Suppression.—A striking phenomenon that appeared in the course of these measurements was the large number of cases that showed a partial or local suppression which takes place only at the fovea.

Of the total number of patients examined 71 per cent showed this type of suppression. It became evident in two different parts of the examination, and has been designated as size suppression and refrac-

tive suppression. In measurements of the size difference, when the patients were asked to look at the light and line on one side of the center circles, they reported the disappearance of either the light, the whole line or part of the line, or both the light and the line. In some cases this disappearance was so persistent that it was impossible for the patient to make any judgment of the relative position of the light and the line. Sixty-four per cent of the cases showed size suppression. The other part of the examination in which this local suppression became evident was in the measurement of the refractive condition. This is done by moving the image of small print backward and forward through the target that is being fixated, until the print comes into sharp focus. In some cases the print usually disappeared just as it came into sharp focus. Fifty-one per cent of the cases showed refractive suppression. Forty-four per cent of the cases showed both size and refractive suppression. After the patient had been given the proper correction, both types of suppression tended to disappear. With some the effect was complete and immediate; with others there was a more gradual improvement. The disappearance of suppression with proper corrections was so universal that its presence or absence was taken as one of the indications for judging the correctness of the prescription given. If suppression persisted, the presumption was that the prescription was not correct.

Different Size Corrections for Distant Vision and Near Vision.—As already stated, the size differences were determined both for 20 feet and for 16 inches (2.50 diopters). In 18 per cent of the cases it was found that the relative size of the ocular images was different at the two distances. Of these eighteen patients, all but two have received relief.

Size Amplitude.—In the examination of certain of the patients the following interesting behavior was observed: Ordinarily, if the size of the image in one eye was changed by 0.25 per cent, a change could be seen in the displacement of the light points relative to the short lines when the patient looked at the size testing targets. However, some of the patients behaved otherwise; as the size of the image in one eye increased, they seemed able to make adjustment of size in the other eye to keep the relative size equal. A typical example was a patient who on first tests showed no size difference. A size increase lens placed before the right eye made the image in the right eye larger than the image in the left eye. When the size increase lens was transferred to the left eye the patient reported equality of size. The interposition of lenses that increased the image in the left eye up to 5.25 per cent caused no apparent change in size. With an increase above 5.25 per cent, however, the patient reported that the image in the left eye was larger than that in the right eye. For correction a 2.25 per cent sphere before the left eye

was given, and some degree of relief was reported. This evidence of size amplitude was shown in 5 per cent of the patients.

Change of Slip with Size.—Another interesting type of behavior shown by certain of the cases was as follows: In measuring the patients, their phoria condition was evidenced by what has been called a "slip," that is, the displacement in the same direction of the points of light relative to the lines. If the points of light that are seen with the right eye are to the left of the lines, there is an exophoria; if the points of light are to the right, there is an esophoria; if the points of light are above, there is a left hyperphoria, and if the points of light are below, there is a right hyperphoria. There were seventeen cases in which slight displacements of this kind changed with the correction of size differences. This was especially evident with a slip in the vertical meridian.

The Effect of Lenses of Incorrect Size.—A corollary to the fact that differences in the relative size of ocular images cause discomfort is the fact that the wearing of size lenses that increase size differences also causes trouble. This was shown in several cases in which incorrect lenses were given. In one obstinate case the patient, after several series of measurements, was given a fitover with meridional size correction for one eye and a zero power flat lens for the other. There was marked relief for two months. Then, without the patient's knowing it, an optician substituted a zero power lens on a 3 diopter base curve for the flat lens. The patient's former symptoms reappeared and lasted until he came to the clinic three weeks later, when the substitution was discovered and corrected. He has since worn the correct prescription for fourteen months with relative comfort. Two other instances might be cited, as well as the fact that normal subjects who wore lenses that increased the size of the image in one eye experienced both ocular discomfort and attacks of headache.

Prisms.—Invariably no prism power was used in the measurements or tests, and none was given in the prescriptions. This was not due to a belief that prisms are never helpful, but because of the distortion of the ocular images produced by them. Prisms have not only the commonly known effect of causing the eyes to change vergence but also the less known effect of distorting the images. This distortion varies with the position, power and base curve of the prism. Due to lack of knowledge as to the effect of such distortion, it seemed undesirable to complicate the problem by introducing it. Of the patients who had prismatic power in their lenses when they came to the clinic, 8 per cent were given prescriptions that included size corrections, but without prisms. The strength of the prisms that these patients had been wearing varied from 0.25 to 4.00 Δ in each eye and averaged about 2.00 Δ.

Four of these patients were among those who obtained marked relief: One had some relief; in one the effect was questionable, and two were in the failure group.

The five who obtained relief all returned for a second test and now wear lenses that were refined after the patients had worn and made adjustment to the first glasses given. The patient in the questionable case and the two in the failure group have been seen once.

STATISTICAL ANALYSIS

This report covers only ninety-six of the total one hundred and twenty referred cases. Of the twenty-four not reported on, seventeen,

TABLE 1.—*Age of Patients (Ninety-Six Cases)*

	Ages Between					
Years	10-20	20-30	30-40	40-50	50-60	60-70
Number	10	25	24	13	19	5
Average age	36.51					
Age of youngest patient	11					
Age of oldest patient	65					

TABLE 2.—*Refractive Condition of the Patients at Twenty Feet (Ninety-Six Cases)*

Refraction	Number	Number with Refraction of 1.50 D. or Less	Average Refraction	Largest Refraction
Emmetropia	11	11	0.00	0.00
Myopia*	26	21	—1.06 D.	—4.25
Hyperopia*	50	40	+1.03 D.	+4.50
Mixed† astigmatism	9	5	—1.00 D. sph. ⊃ +2.22 cyl.	—1.50 D. sph. ⊃ +4.25 cyl.
Total	96	77		

* Average refraction includes spheres and cylinders for two eyes.
† Cases in which spheres and cylinders would not reduce wholly to plus or minus.

although they showed size differences, were examined before the size lenses were available, and so they were not given size corrections. In the remaining seven cases, either no size difference was found or sufficient data were lacking.

Age, Refraction and Phoria.—In order to determine the type of patient that was dealt with, tables giving the ages, refractive condition and phorias were compiled (tables 1, 2, 3 and 4). Table 5 gives the relation between the refractive and horizontal phoria condition.

A study of these tables shows that the patients examined represented an average group. The ages ranged from 11 to 65 years, averaging 36 years. As to refraction, there were eleven emmetropes, twenty-six myopes with an average error of — 1.06 diopters; fifty hyperopes with an average error of + 1.03 diopters, and nine patients having mixed astigmatism with refraction averaging — 1.00 diopter sphere + 2.25 diopter cylinder. As to phoria, five were orthophoric, thirty-seven were

exophoric with an average of 1.82 Δ and thirty-nine were esophoric with an average of 2.43 Δ.

Symptoms.—As would be expected, most of these patients showed marked symptoms. For purposes of analysis, these symptoms were divided into two groups: (1) general and (2) ocular. These data are given in tables 6 and 7. Data on the duration of symptoms are given in table 8.

TABLE 3.—*Horizontal Muscle Condition of Patients (Eighty-Three Cases)* *

Muscle Condition	Number	No. with Phoria: Of 1.5 Δ or Less at 20 Ft.	No. with Phoria: Of 5 Δ or Less at 2.5 D.	Average Phoria, 20 Ft.	Average Phoria, 2.50 D	Largest Phoria, 20 Ft.	Largest Phoria, 2.50 D.
Orthophoria	7	7	..	0	0	0	0
Exophoria	37	23	22	1.81Δ	*5.99Δ	8.0Δ	15.0Δ
Esophoria	39	22	9	2.43Δ	*2.68Δ	12.0Δ	12.0Δ
Total	83	52	31				

* In the 96 cases, horizontal phoria was determined at 20 feet for 83 cases and at 16 inches (2.50 diopters) for 59 cases.

TABLE 4.—*Vertical Phoria and Cyclophoria of Patients at Twenty Feet*

Muscle Condition	Number	Number with Phoria of 1Δ or Less	Average Phoria	Largest Phoria
Vertical phoria	53	51	0.45Δ	4.60Δ
Cyclophoria	4	..	1.50°	1.50°

TABLE 5.—*Relation Between the Refractive Condition and Muscle Condition of Patients at Twenty Feet (Ninety-Six Cases)* *

Refraction	Number	Number with Orthophoria	Number with Exophoria	Number with Esophoria
Emmetropia	11	1	6	4
Myopia	26	3	17	6
Hyperopia	50	3	19	28
Mixed astigmatism	9	0	5	4
Total	96	7	47	42

* Thirteen of these patients did not have actual phoria readings recorded in the laboratory.

A study of the data on general symptoms shows that eighty of the ninety-six had one or more general symptoms. Seventy-eight had either headaches or stomach trouble.

A study of the data on ocular symptoms shows that ninety-one of the ninety-six had one or more ocular symptoms, fifty-six had photophobia and seventy-seven had eyes that tired, burned or pained. Sixty-four reported trouble from motion pictures or from automobile or train riding.

Type and Amount of Size Differences.—From the accompanying tables a good idea may be obtained of the type of case in which the size

differences have been determined. The kind and amount of size differences found, together with the refractive condition, are given in table 9. The amount of size differences found in the clinical cases has little significance without a knowledge of the size differences existing in normal persons. To obtain some idea as to what this might be, measure-

TABLE 6.—*General Symptoms of Patients (Ninety-Six Cases)*

Number with Some General Symptom	Number with Headaches	Number with Stomach Trouble	Number with Dizziness	Number with "Tight Band Around Head"	Number Who Reported Nervous Conditions	Number Who Reported Nervous Breakdown
80	75	30	12	3	8	9

Of the 96 patients: 78 had either headaches or stomach trouble
27 had both headaches and stomach trouble
5 reported general symptoms only
17 reported ocular symptoms only
74 reported both ocular and general symptoms

TABLE 7.—*Ocular Symptoms of Patients (Ninety-Six Cases)*

	Number with Some Ocular Symptom	Photophobia	Eyes Tired	Eyes Burned	Eyes Ached; Pain	Feeling of Tension in Eye	Eyes Blurred	Trouble in Near Vision	Trouble at Movies	Trouble in Automobile or Train	Sleepiness
Number.....	91	56	53	40	42	16	31	40	49	57	20

64 reported trouble either at moving pictures or while riding in an automobile (troubled by moving objects)
42 reported trouble both at moving pictures and while riding in an automobile
5 reported general symptoms only
16 reported ocular symptoms only
73 reported both ocular and general symptoms

TABLE 8.—*Duration of Symptoms (Ninety-Six Cases)*

	Duration						
	Less Than 1 Year	From 1-5 Years	5-10 Years	10-20 Years	20-30 Years	30-40 Years	40-50 Years
Number.....	5	39	19	13	9	5	6

Average duration ..... 10.07 years
Shortest duration ..... 6 months
Longest duration ..... 46 years

ments were made in sixteen persons who were having no ocular trouble. While this is too small a number from which to assume that the averages obtained represent a true normal, it is sufficient to give valuable indications of what the normal may be.

Table 10 gives the findings in this group. Table 11 gives the refractive condition of this group. Comparing tables 9 and 10, it will be seen that there were six normal persons who had no size difference; that the average overall size difference of the normal persons

who had such a difference was 0.82 per cent (average for near and far) as compared with 1.13 per cent in the clinical cases, and that the average meridional size difference of the normal persons was 0.79 per cent as compared with 1.90 per cent for the clinical cases.

Clinical Effect of Correction of Size Difference.—The determination of the clinical effect of the correction of the size differences was based on the relief of symptoms, both general and ocular, over a sufficiently long period to insure its permanency.

TABLE 9.—*Size Condition of Patients at Twenty Feet (Ninety-Six Cases)*

Type of Size Condition	Number	Average Size Difference Between Eyes, per Cent	Largest Size Difference, per Cent
Spherical	44	1.13	3.00
Cylindric	31	1.90	4.25
Spherocylindric	21	0.84 sph. ⌒ 1.04 cyl.	1.62 sph. ⌒ 1.75 cyl.

TABLE 10.—*Size Condition of Normal Persons (Sixteen Cases)*

	Number		Average Size Difference Between Eyes, per Cent		Largest Size Difference, per Cent	
Type of Size Condition	20 Ft.	2.50 D.	20 Ft.	2.50 D.	20 Ft.	2.50 D.
No size difference	6	6	0.00	0.00	....	....
Overall	6	9	0.83	0.81	1.25	1.25
Meridional	3	1	0.83	0.75	1.00	0.75
Overall-meridional	1	0	0.50 sph. ⌒ 0.50 cyl.	....	....	....

TABLE 11.—*Refractive Condition of Normal Persons (Sixteen Cases)*

Average Refraction at 20 Ft.		Average Refraction at 2.50 D.		Visual Acuity at 20 Ft.			Visual Acuity at 2.50 D.		
Left Eye	Right Eye	Left Eye	Right Eye	Right Eye	Left Eye	Both	Right Eye	Left Eye	Both
0.48 D.	0.49 D.	2.47 D.	2.47 D.	20/17	20/18	20/14	20/16	20/17	20/14

The length of time that the size corrections have been worn is shown in table 12. The longest period is thirty-seven months; the average fifteen and six-tenths months and the shortest four months. Only ten of the seventy-six patients who received relief have worn their corrections less than six months.

Close touch has been kept with all the patients, and from examinations and reports it is considered that of the total of ninety-six patients twenty-one have received complete relief, fifty-five partial relief and twenty no relief.

A study of the characteristics of the cases in the three groups has been made in an attempt to determine the reason for the difference in the relief obtained.

The characteristics considered are: (1) age, refraction and phoria; (2) symptoms, and (3) type and amount of size difference. The averages of these characteristics for the three groups are given in table 13.

A study of table 13 shows that the characteristics in the three groups did not differ essentially. In type of symptoms, average refraction, phoria and size difference there was a marked similarity. The only difference that appeared was that the average horizontal phoria of the complete relief group was less than half the horizontal phoria of the other groups.

A possible explanation for the lack of success in the unrelieved cases is that at the present time clinical work is confined to symmetrical overall or meridional size differences. Besides differences of this type there exist other types, such as asymmetric or distorted size difference. Research on the effect of such size differences is in progress. In eleven

TABLE 12.—*Length of Time the Size Corrections Have Been Worn (Seventy-Six Cases)* *

	Patients Now Wearing Size Correction	Patients Who Have Worn Size Correction			
		6 Months or Less	6-12 Months	12-18 Months	More Than 18 Months
Number.....	76	10	23	15	28

Average length of time that size correction has been worn	15.64	months
Shortest time that size correction has been worn	4	months
Longest time that size correction has been worn	37	months

* Twenty of the 96 cases reported were failures and so in most cases these patients are not wearing a size correction.

of the twenty cases in which treatment was a failure the patients have been to the clinic only once. There is evidence that as the patients wear the prescribed lenses, a readjustment occurs and there is frequently a change of refraction and size with a consequent need for refinement of the correction. Whether or not some measure of relief could have been given in these cases by further tests is an open question. In the twenty cases listed as failures there are five patients who have been to the clinic several times and who, admittedly, have particularly obstinate cases. Three of these five have a large exophoric slip at near vision. The fourth patient, aged 27, who has been examined thoroughly, has been under observation by a noted brain specialist and has had eleven prescriptions from six ophthalmologists in the past eight years. These ranged from — 0.25 diopter cylinder axis 180 to + 0.50 diopter cylinder axis 90 in each eye and from + 0.50 diopter sphere to + 0.87 diopter sphere. He has also had various combinations of these powers. Headaches and eye discomfort still persist. The fifth patient has headache and stomach involvement. She is being treated for the latter, but her

headaches have not diminished. There is a large amount of suppression manifested, which would indicate some degree of ocular trouble. With further research, it is hoped that means may be found to give relief in these cases.

TABLE 13.—*Averages*

	Total Group (96 Cases)	Group Showing Complete Relief (21 Cases)	Group Showing Partial Relief (55 Cases)	Group Showing No Relief (20 Cases)
Average age	36.5	40.1	36.5	32.8
Refractive condition:				
No. of cases of emmetropia	11	2	7	2
No. of cases of myopia	26	4	17	5
No. of cases of hyperopia	50	12	27	11
No. of mixed cases	9	3	4	2
Average refraction in				
Cases of myopia	−1.06 D.	−1.49 D.	−0.97	−1.01
Cases of hyperopia	+1.03 D.	+0.88 D.	+1.00	+1.28
Mixed cases	−1.00 D. sph. ⊃ +2.25 cyl.	−1.66 D. sph. ⊃ +2.91 cyl.	−0.85 D. sph. ⊃ +1.81 cyl.	−0.68 D. sph. ⊃ +1.25 cyl.
Average error of group	1.03 D.	1.20 D.	0.92 D.	1.08 D.
Phoria condition:				
(a) Horizontal (measurements in 83 cases only):				
No. with orthophoria	7	2	5	0
No. with exophoria	37	6	21	10
No. with esophoria	39	10	20	9
Average value of				
Exophoria at 20 ft	1.81△	1.17△	1.70△	2.44△
Esophoria	2.43△	0.95△	3.46△	1.77△
Horizontal phoria of group	1.95△	0.92△	2.28△	2.12△
(b) Vertical (measurements in 53 cases only):				
No. with vertical phoria	53	15	24	14
Average value of vertical phoria	0.45△	0.56△	0.42△	0.37△
Symptoms:				
Number of patients with				
General symptoms	80	17	46	17
Ocular symptoms	91	21	50	20
Both general and ocular symptoms	74	17	40	17
General symptoms:				
(1) Headaches	75	16	42	17
(2) Stomach trouble	30	4	20	6
Ocular symptoms:				
(1) Eyes tired	53	12	32	9
(2) Eyes burned	40	11	21	8
(3) Eyes pained and ached	42	10	24	8
(4) Eyes blurred	31	8	17	6
(5) Photophobia	56	15	30	11
Suppression	71	16	38	17
Type and amount of size differences:				
Number with correction				
Overall	44	11	24	9
Meridional	31	7	19	5
Overall-meridional	21	3	12	6
Average size difference between eyes				
Overall, per cent	1.13	1.36	1.00	1.21
Meridional, per cent	1.90	2.18	1.70	2.30
Overall-meridional, per cent	0.84 sph. ⊃ 1.04 cyl.	0.73 sph. ⊃ 1.18 cyl.	0.95 sph. ⊃ 1.03 cyl.	0.66 sph. ⊃ 1.00 cyl.
Of group, per cent	1.54	1.71	1.46	1.62

Headaches, Photophobia and Suppression.—The large number of patients reporting headache, photophobia and suppression, the neurologic nature of these symptoms and the fact that both headache and suppression and often photophobia disappeared with size corrections suggest that a statistical study of these symptoms would be of interest. Table

14 gives the data arranged to show the correlation between these symptoms. Of the ninety-six patients seventy-five had headaches, fifty-six photophobia and seventy-one suppression. Of the seventy-five patients with headache sixty-eight had either photophobia or suppression. Of the fifty-six patients with photophobia, fifty-four had either suppression or headaches. Of the seventy-one patients with suppression, sixty-six had either headaches or photophobia.

REPORT OF CASES

As examples of the type and treatment of patients who received complete relief from their corrections, five case reports will be given.

CASE 1.—Mr. H. R. M., aged 54, a business man, was wearing: for distance vision, right eye, +0.50 diopter cylinder axis 135; left eye, +0.25 diopter sphere +0.25 diopter cylinder axis 70, with +1.75 diopter sphere added for each eye for near vision.

TABLE 14.—*Number of Patients with Photophobia, Suppression and Headaches (Ninety-Six Cases)*

	Headaches	Photophobia	Suppression
Photophobia	46	56	44
Suppression	58	44	71
Headaches	75	46	58

Of the 75 patients with headaches, 68 had either photophobia or suppression
Of the 56 patients with photophobia, 54 had either headaches or suppression
Of the 71 patients with suppression, 66 had either headaches or photophobia
Thirty-six of the patients had photophobia, suppression and headaches

For three years prior to his examination at the clinic on Aug. 5, 1930, this patient had constant left temporal headaches and a feeling of pressure back of the left eye. He also complained of pain back of the eyes when he was nervously excited or when he coughed or sneezed. He had been forced to limit his reading and could not read by artificial light without a marked increase of discomfort. Several complete examinations by a number of physicians had not disclosed the reason for these symptoms. His upper teeth, as a possible source of infection, had been extracted, but no change in his condition was noted.

The foregoing prescription was given the patient on March 29, 1930. His refractive error was relatively small. His muscle imbalance was 1.00 Δ exophoria for distance and 6.00 Δ exophoria for near. Refraction with the prescription given showed that the left eye was focused at 20 feet, but that the right eye was focused about 0.12 diopter outside 20 feet, that is, the right eye was 0.12 diopter hyperopic.

With this refractive condition the right eye ocular image vertically was larger than the left eye ocular image vertically. There was refractive and phoria suppression and unsteadiness of images. As correction of size and refraction proceeded, an overall size difference was manifested. Data at reading distance were difficult to obtain because the patient lost fusion. The patient was given for trial the equivalent of: right eye, + 0.50 diopter sphere + 0.50 diopter cylinder axis 135 (flat); left eye, + 0.62 diopter sphere + 0.25 diopter cylinder axis 70 (9 base, a 1.20 per cent increase), with + 1.75 diopter sphere added for each eye.

One week later, tests showed that refraction was still balanced, but that size was overcorrected by 0.40 per cent. There was no refractive suppression, the images were steadier and measurements at reading distance were possible. The only change in the prescription was in the curvature of the left lens. The patient was given: right eye, +0.50 diopter sphere +0.50 diopter cylinder axis 135 (flat); left eye, +0.62 diopter sphere +0.37 diopter cylinder axis 70 (6 base, a 0.80 per cent increase), with +1.75 diopter sphere added for each eye.

The patient wore this on a business trip to Europe. He returned for a recheck on December 12. He reported that at first there was no improvement in his condition. Later there was a gradual change and he was free from headache on an average of two days per week.

At this time a further change in refraction and size was made. The right eye image was larger overall. Increasing the left eye image 0.80 per cent to correct this difference, it was necessary to reduce the refractive sphere for the left eye by 0.12 diopter. Thus the prescription given finally was the equivalent of: right eye, +0.50 diopter sphere +0.50 diopter cylinder axis 135 (flat); left eye, +0.50 diopter sphere +0.25 diopter cylinder axis 70 (12 base, a 1.60 per cent increase), with +1.75 diopter sphere added.

Four months later the patient reported that he was no longer forced to limit his reading, and that he had no headache or feeling of pressure back of the eyes.

In general terms, his condition was: 1. The image in the right eye was larger overall than the image in the left. 2. The focus of the right eye was hyperopic relative to the focus of the left eye. A plus sphere added to the right eye equalized refraction, but made the image in the right eye still larger. A size increase added to the left eye equalized size. The size correction was an overall increase in one eye.

CASE 2.—Mrs. K., aged 23, a housewife, was wearing: right eye, —4.00 diopter sphere +0.25 diopter cylinder axis 180; left eye, —4.75 diopter sphere +0.50 diopter cylinder axis 180.

This patient had been forced to give up practically all reading. At near, her eyes ached and were easily fatigued; vision blurred and images were unsteady. She complained also of constant photophobia and occipital headaches.

While in college she had been forced to limit her work and to follow a prescribed routine in the use of her eyes. After graduation she accepted a position as reader in economics, hoping that work under much less pressure than that required in college would enable her to use her eyes with less discomfort. She had to resign after two days. Her refraction was checked on Sept. 23, 1930, and no change was made.

She had an emotional temperament, and the impression was that she was neurotic. She had been given a psychologic test for neurosis and was found to be normal. She was encouraged to use her eyes, but her trouble still persisted.

She was examined at the clinic on December 5. With the aforementioned prescription, given her in 1928, she was balanced refractively to within 0.12 diopter. When relative size was tested, it was found that the right eye image was larger than the left eye image. There was suppression in the left eye during refractive measurements. A 1.75 per cent overall increase of the left eye image made it the same size as the right eye image horizontally and practically the same size vertically. A zero power lens on a 12 diopter base curve which increased the image size 1.60 per cent was placed over the left eye lens prescribed in 1928.

She was now able to read all she wanted to. Her photophobia had lessened so that she was able to go skiing. It is interesting to note also that she formerly complained of a reddish tinge in objects that she saw. Fuchs said that this condition of erythropia occurs in normal eyes that have been exposed to a very bright light and particularly in aphakic patients. The erythropia disappeared.

In this case the condition was difficulty in using the eyes for near vision. The refractive correction was not changed. Mental therapy was used, to no avail. The only correction given was an overall size increase of 1.60 per cent in one eye.

CASE 3.—J. W. McC., aged 32, an insurance broker, was wearing: right eye, +0.62 diopter sphere, and left eye, +0.62 diopter sphere +0.12 diopter cylinder axis 180, for reading only.

For a period of eleven years this patient had experienced considerable discomfort in reading. He complained that his eyes pained and tired markedly at near. There was also a burning sensation in the eyes and photophobia.

In 1920, he had been operated on for 12.00 △ of esophoria, and despite the reduction of phoria, his discomfort still continued.

He came to the clinic and was tested for refraction and size difference on Nov. 15, 1929, shortly after the special lenses used for size measurements had been obtained and before a method of coordinating refraction and size had been evolved. Fading and suppression of images was evident. Without lenses his eyes were balanced at 20 feet and were focused slightly outside of 2.50 diopters at near but within 0.12 diopter of each other. At distance, he was 0.33 △ exophoric and at near, 3.00 △ exophoric. He was given at this time for distance: right eye, zero power lens (6 base, a 0.80 per cent increase), and left eye, zero power lens (flat); for near vision: right eye, 2.00 per cent cylinder axis 180; left eye, 1.50 per cent cylinder axis 90.

With this he was comparatively comfortable for several months. He was rechecked on Aug. 20 and Sept. 8, 1930. At this time the size correction for the right eye at near given him the previous November manifested itself for distance. Refractively the left eye was more hyperopic than the right eye. The suppression noted during his first tests had decidedly decreased. He was given for distance: right eye, 2.25 per cent cylinder axis 180, and left eye, + 0.25 diopter sphere + 0.37 diopter cylinder axis 180, with a size addition of 2.00 per cent cylinder axis 90 for near vision.

He has been wearing this correction for a year and has had practically complete relief. His eyes tire some after a few hours of close work, but without the prescription he cannot read at all. Putting the extra lens on for reading proved to be somewhat bothersome and he tried to work without it, as he afterward reported, but his original trouble quickly returned.

In this case, there is quite definite ocular trouble with a very small refractive error and phoria. The trouble is out of proportion to the defects. Here also the relative size at distance differs from that at near vision and the correction is in one meridian.

CASE 4.—Mr. L., aged 49, a professional man, was wearing: right eye, + 0.87 diopter sphere + 0.25 diopter cylinder axis 80; left eye, + 0.87 diopter sphere + 0.25 diopter cylinder axis 100, with + 2.25 diopter sphere added.

Since 1920, this patient had been having a great deal of difficulty with near vision. Reading for ten or fifteen minutes resulted in headaches; reading for longer periods brought on headaches which sometimes lasted several days. He had been forced to give up work entirely and to confine his reading to his personal correspondence. Examination of his eyes by several very reputable ophthalmologists failed to disclose any ocular defects, and a thorough general examination did not reveal any apparent basis for his trouble.

He came to the research department on Dec. 8, 1926. Measurements at 20 feet showed that he had 2.5 degrees of excyclophoria, which increased to 3.5 degrees at reading distance. At 20 feet he was just within the limit of his cyclofusional amplitude. At 16 inches, however, he was outside his amplitude and so was not able to fuse horizontal diameters at that distance.

About a year later an optical device was developed by means of which it was possible to rotate one of the patient's visual fields and so to correct his excyclophoria.[4]

With this spectacle device he was able to read very comfortably from two to three hours continuously, without any headache. This lasted until late in 1930, when the patient's eyes began to bother him again.

He came to the clinic on March 2, 1931. Examination of the field rotating device at this time showed that it was decidedly out of adjustment, so that a large horizontal phoria was introduced. This might more than account for the patient's symptoms at this time. During the examination it was found that he had a size difference.

He was given the following prescription: for distance, right eye, + 0.75 diopter sphere + 0.25 diopter cylinder axis 80 (flat) ⊃ 0.75 per cent cylinder axis 180, and left eye, + 0.87 diopter sphere + 0.25 diopter cylinder axis 100 (6 base); for near, right eye, + 2.75 diopter sphere + 0.25 diopter cylinder axis 80 (flat), and left eye, + 2.75 diopter sphere + 0.25 diopter cylinder axis 100 (6 base) ⊃ 0.50 per cent cylinder axis 180.

This correction was substituted for the field rotating device for reading. Six weeks later the patient reported entire success with the new reading glasses, though there was some indication of eye ache following the use of the distance glasses at golf, etc. Four and a half months later he reported this correction a "great success."

Case 5.—Mr. H. C. R., aged 22, a business man, was wearing: right eye, + 0.75 diopter sphere + 0.50 diopter cylinder axis 100; left eye, + 1.00 diopter sphere + 0.62 diopter cylinder axis 70.

This patient had no serious trouble until his senior year in college. At that time he began to have trouble studying, and his general condition was also affected. He was having general headaches daily, with some localization temporally. He was a member of the track team, but was apparently unable to stand the hard training, and had had a fainting spell just before a track meet. He underwent a thorough examination, with negative results, and was sent to the clinic on March 19, 1929, to have his eyes checked. He had constantly worn glasses since he was 8 years old. The correction that he was wearing showed an undercorrection of the astigmatism in the left eye and an overcorrection in the right eye. Examination showed that he had between 0.25 and 0.50 diopter of astigmatism in the right eye and about 2 diopters of astigmatism in the left eye. When he was measured without any glasses his images were very nearly the same size, but with the astigmatic correction there was a meridional size difference.

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4. Lancaster, W. B.: The Ames Spectacle Device for the Treatment of Cyclophoria, with a Report of a Successful Case, Arch. Ophth. **57**:332 (July) 1928.

As the meridional size lenses had not been developed at that time, no prescription could be given him that corrected both his refractive and his size errors. It was suggested that he go without his glasses and see how he got along. He reported, several months later, that there was a marked improvement in his general condition. He was able to pass his examinations and finish college without any correction for his eyes. This indicated that he could more easily carry an astigmatic error than a size difference.

The patient got along well the first part of the summer, but began to have trouble about July. He returned to his ophthalmologist, who gave him the following refractive correction: right eye, + 0.75 diopter sphere + 0.62 diopter cylinder axis 90; left eye, + 1.00 diopter sphere + 1.75 diopter cylinder axis 70. This worked well for about two weeks and gave him marked relief from his trouble. At the end of that time, however, he began to have difficulty again. He then found that he had trouble whether he wore his glasses or went without them. He came to the clinic again on November 16, wearing the following correction: right eye, + 0.75 diopter sphere + 0.62 diopter cylinder axis 90; left eye + 1.00 diopter sphere + 1.75 diopter cylinder axis 70. The refraction was slightly hyperopic. The phoria record was 1.20 △ esophoria for distance and 1.80 △ esophoria for near. There was marked suppression. By means of the size cylinders, which had been developed since his first visit, it was now possible to correct both his astigmatism and size differences. The size correction that best balanced his images was found to be a 3.25 per cent cylinder axis 70 in the right eye. The correction given him experimentally was: right eye, 3.25 per cent cylinder axis 70; left eye, + 2.00 diopter cylinder axis 70.

A month later he was reading an hour a day, whereas he had done practically no reading previously. There had been almost immediate lessening of photophobia. His headaches had decreased, but he was still bothered by them. Later in the winter the glare from snow bothered him, and train riding particularly caused an upset.

He was rechecked on March 10, 1930, just prior to his accepting a position with a corporation. Because he was relatively comfortable, no change was made. The suppression that was so marked during his previous tests was practically absent. Later, he was able to carry on his office work, but could do no recreational reading.

In April, 1931, he was transferred to an accounting department. After three weeks, he noticed that lights bothered him; his eyes felt tense, and he had frontal headaches and blepharitis. He was rechecked on May 18, and both refraction and size were found to be unbalanced. On the basis of measurements made, he was given: right eye, + 0.50 diopter cylinder axis 90 ⊃ 0.50 per cent cylinder axis 90; left eye, +2.00 diopter cylinder axis 70 ⊃ 1.00 per cent cylinder axis 180.

He returned to the accounting work that had troubled him before, and his reports indicate that he is now having relatively little trouble. There has been a marked change in his health and in his general attitude toward life.

SUMMARY AND CONCLUSION

A summary of the foregoing data would indicate the following:

1. Differences in the size of the ocular images of the two eyes may exist without causing discomfort, but in such cases the differences are less than when there is discomfort (tables 9 and 10).

2. Differences in the size of the ocular images associated with discomfort are found in persons of all ages (table 1) with emmetropia, all types of refractive errors (table 2) and orthophoria as well as with all types of heterophoria (table 3).

3. The symptoms associated with a difference in the size of the ocular images are of two types: first, those of a general type such as headache and stomach disorder, apparently involving the sympathetic nervous system (table 14); second, those of a localized ocular type, such as photophobia, suppression, tiring, burning, tension of the eyes and disabilities, such as difficulty in using the eyes in reading, automobile or train riding, etc. (table 7).

4. Correction of size differences apparently brought complete relief in about 20 per cent of such cases, partial relief in 60 per cent and no relief in 20 per cent (table 13).

In reference to paragraph 1, it would be expected that certain amounts of size differences may exist with no discomfort, just as it is known that in many cases refractive errors may be present without causing trouble. The fact that no size differences of more than about 5 per cent were found in all the patients examined would seem to be significant. It would suggest that a difference of about 5 per cent is the limit of size difference that can be tolerated; that is, if a size difference beyond that amount is present, either complete suppression or diplopia takes place. There may be some connection between this figure and the largest amount of anisometropia that can safely be corrected, which is about from 1.5 to 2 diopters. The change of size produced by a $\pm$ 2 diopter lens is about 4 per cent.

In reference to paragraph 2, the number of cases showing large refractive and phoria errors is low compared with the number of cases showing small errors (tables 2 and 3). The explanation may be that there has not been so much inclination on the part of doctors to refer those patients who have marked physiologic defects that might account for their difficulties.

In reference to paragraph 3, it should be borne in mind that the locus of the beginning of the excitation in the case of size differences is in the occipital lobes of the brain, where the two ocular-images are fused, and not in the eyes themselves. The brain manifestation in these cases is well shown in the frequency and correlation of photophobia, suppression and headaches (table 14).

It is not surprising, therefore, that this type of patient should have brain disturbance and general disturbances of the sympathetic nervous system, such as the symptoms disclose. Nor is it surprising that in many of this group the diagnosis is "neurotic," especially in view of the fact that up to the present apparently no physiologic basis existed for the symptoms of which they complained.

The presence of ocular symptoms, such as tiring of the eyes, burning and tension, would naturally follow from an ocular effort to correct the size differences that manifest themselves in the occipital lobes. There are numerous motor nerve fibers running from the lobes to the eyes, some direct and some through the nuclei. Their ocular terminals are in the extrinsic muscles, the ciliary body and the retina.

In reference to paragraph 4, the field of investigation of size differences has barely been opened. The symmetrical size differences overall, or in one meridian, that have been determined and corrected represent but one type of difference in the size and shape of the ocular images. This may possibly account for the failure to give relief in some cases. Several interesting fields await further investigation, such as the effect of asymmetric size differences on vision, the effect of changing the size of the images in both eyes and the correlation between size differences with near and distant vision. The question as to whether size differences are a factor in particular ocular defects, such as progressive myopia, about which one seems to know so little, offers interesting studies.

It might seem at first thought that the number of cases requiring such an elaborate examination is too limited to warrant the procedure. It is felt as the work goes on that there are many patients getting only partial relief from other therapeutic measures who might show, on such an examination, size differences of a disturbing amount. It is, of course, impossible to form any definite opinion as to the percentage of the number of cases of this type. Our own personal experience is as follows: With a daily number of cases varying from two to ten, we see five or six patients a week who, we feel more or less certain, have size anomalies. Beyond this number, we see many cases about which we formerly felt satisfied as to our diagnosis, but about which we now hesitate to form a definite opinion until the size defects have been determined.

The demonstration that the relative size and shape of the ocular images play an important part in the functioning of the eyes imposes on members of the medical profession the responsibility of determining the possible clinical effect of anomalies in this field. It was for this very purpose that the clinical investigations here described were undertaken. The evidence presented that the existence of this new type of physiologic defect may be the cause of various ocular troubles would seem to show that the ordinary methods of measurement do not disclose all the abnormal physiologic conditions that may exist, and that, at least in all doubtful cases, too definite conclusions should not be formed until measurements have been taken to determine the relative size and shape of the ocular images.

HYPERPHORIA

DIAGNOSIS AND TREATMENT

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In no branch of muscle work is there as much uncertainty and confusion, both as to diagnosis and as to treatment, as in the subject of hyperphoria. Diagnoses vary as widely as the test by which the diagnoses are made, and the treatment varies still more widely. These tests and treatments have all been successful part of the time; else they would not be persisted in, but the chief difficulty seems to be in the proper use of the tests, the correct interpretation of the findings, the correct diagnosis and the best choice of treatment for the particular case at hand. The common mistake is to have a more or less pet plan of procedure and to make each case conform to this plan.

A very common statement in textbooks is to correct two thirds of the hyperphoria by prisms; for example, if the phorometer shows 6° of hyperphoria for distance, 4° according to this rule is well borne. The reason why this so frequently fails will be referred to later.

The difference in the hyperphoria for distance and for near has been pointed out, but the reason for this and how it would modify treatment has been rather neglected.

It is unwise to change nomenclature too often, but, when with a slight change in interpretation a difficult problem may be simplified, this variation seems justifiable. My reference is to the terms hyperphoria and hypophoria, and hypertropia and hypotropia.

When speaking of hyperphoria as opposed to hypophoria, one is saying that the hyperphoric eye is higher than it should be and that the hypophoric eye is lower than it should be. But these terms are being used before it has been proved which is correct. In my experience, at least 98 per cent of cases showing a difference in level of the eyes are due to a weakness of one or more of the elevators or depressors, with usually some secondary contraction of the direct antagonist and secondary deviation of the associate antagonist. Nothing can be intelligently done in the diagnosis and treatment in such a case until the offending muscles are determined. Then, instead of a diagnosis of right hypophoria, one has a diagnosis, for example, of paresis of the

Submitted for publication, Dec. 8, 1932.

The paper was read on Oct. 19, 1931, before the Section on Ophthalmology of the Newark Academy of Medicine.

right superior rectus. This tells at once that the right eye is lower than it should be. But the cause of this is not known until the paretic muscle is determined. When this fact is learned, neither the term hyperphoria or hypophoria is wanted, but the diagnosis is given by naming the affected muscle.

For simplicity in working and teaching, I do not use the terms hypophoria or hypotropia but right and left hyperphoria or hypertropia. This does not commit me to an opinion until I have made the diagnosis; then the affected muscle is stated in the diagnosis as paresis of the right superior rectus, right superior oblique, etc. After using this simplification for some years in teaching at the New York Post-Graduate Medical School and Hospital and elsewhere, the results with untrained men have been very satisfactory. More mistakes are made by the experienced men, who continue to use these terms as opposed to each other.

The amount of the hyperphoria is no indication of the amount of trouble it can give. Slight amounts may be the source of marked symptoms, while decidedly larger amounts may be entirely without symptoms. The amount, too, has but little bearing on whether the deviation is a hyperphoria or a hypertropia, as slight amounts are often the latter, while larger amounts are overcome in one way or another. The normal function of sursumvergence no doubt helps overcome small amounts of hyperphoria, while a head tilt will correct marked differences in level.

The difference between hyperphoria for distance and near has often been noted. The explanation of this, I believe, is as follows: The majority of cases of hyperphoria are due to a paresis of a superior or an inferior rectus muscle, the superior oblique and inferior oblique muscles being much less frequently affected and in the order mentioned. When measured at 20 feet, the eyes are in the eyes front position, in which position the vertical recti are the main elevators or depressors. At the distance of from 25 to 35 cm. the oblique muscles are the main elevators and depressors, and hence, when the superior or inferior rectus is paretic, hyperphoria is greater for distance and less for near. However, in paralysis of a superior oblique or an inferior oblique muscle, hyperphoria is found to be more for near than for distance. This may not be so, however, if a marked secondary deviation is present. These points must be considered when prescribing prisms for relief of hyperphoria, as will be spoken of later.

Double hyperphoria is a condition much more commonly seen than would be indicated by the literature or the importance it seems to carry in textbooks. The terms anaphoria and cataphoria have tended only to place the condition in the more or less unexplainable class and have led to confusion. All cases of double hyperphoria that I have seen are due to paresis more or less marked of the same muscle in each eye.

These are found in the following order: A large majority of the cases studied have been due to paralysis of both superior recti; next, of both inferior recti, and much less frequently, of both inferior oblique muscles. Paralysis of both superior oblique muscles has not been observed by me. When the muscles are equally paretic, the right hyperphoria will equal the left, but if, for example, the right superior rectus is the more paretic, the left hyperphoria will be greater than the right. Cases are also seen in which left hyperphoria predominates, and there is no right hyperphoria until the eyes are directed up and left, when the left superior rectus is found to be paretic also, with the accompanying increase in right hyperphoria in this field. Any other pair of paretic muscles may have similar findings. Measurements of the deviation in the upper right and left and the lower right and left corners will determine the affected muscles.

To measure double hyperphoria in the primary position, I use the screen test with the prism before first one eye and then the other, supplemented by the Maddox rod or screen-Maddox rod before first one eye and then the other.

The prolonged occlusion test has shown a variation in the kind and amount of hyperphoria. In my experience and belief, it is in these cases of double hyperphoria that this occurs, and it is definitely determined by the methods described.

The relation of hyperphoria to exophoria and esophoria has been variously stated. The two conditions may be brought about independently, but more often the esophoria or exophoria are a result of hyperphoria. One evident proof of this is seen in the reduction of an esophoria or exophoria when the hyperphoria is corrected by prism or operation.

When operation is indicated, if this difference is found in testing either a hyperphoria or a hypertropia, it is better to correct the difference in level before attacking the lateral muscles. There are exceptions to this rule, but with the findings given this is the better procedure. In hypertropia, when correction of the vertical deviation by prism influences but little the lateral, then the lateral deviation may safely be corrected first.

TESTS

The tests used first are the screen and parallax. A deviation of less than 0.5 Δ may be observed by the former, while an observing patient will note an amount as low as this in the parallax test. These tests are made for distance and near in the eyes front position.

Next the deviation is measured in the six cardinal directions (fig. 1).

In most instances this will determine definitely the paretic muscle or muscles.

Primary overacting muscles are rarely seen, but they frequently overact due to a secondary deviation or a secondary contraction.

As a further check, I use the screen-Maddox rod test as described by Maddox and later by Dolman. Place the Maddox rod before one eye and find the amount of deviation at 20 feet. Then cover and uncover the eye slowly, placing prisms before the eye until the line of the rod crosses the spot light when the eye is first uncovered. It is usually found that the hyperphoria increases as the test is continued, and when the screen is left off, that the deviation becomes less and less. This is not always so, but is a very frequent observation and gives an idea of the patient's ability to overcome his hyperphoria.

The tangent curtain is used to map out the field of diplopia, after the foregoing tests are made; this is rarely necessary for a diagnosis. It is, however, further confirmation and is an important record to compare with later tests made to show the effect of treatment, whether it is operative or otherwise.

The common directions for use of the Maddox rod is to use it as a routine measure before the same eye, but much is lost if this is adhered to. It is often possible to demonstrate only double hyperphoria in the primary position by placing the Maddox rod before first one eye and then the other. Primary and secondary deviations are also usually brought out best by this method, primary deviation being shown when the prism is before the paretic eye, and secondary deviation when placed before the sound eye. The primary and secondary deviations may

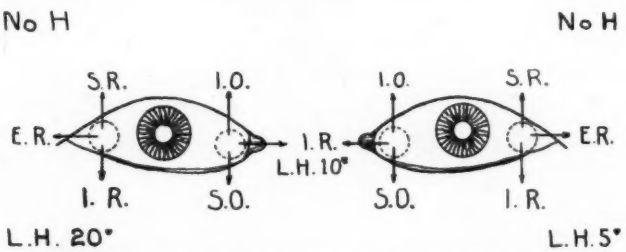

Fig. 1.—Left hyperphoria of 10 △ as shown in the primary position which decreases to 0 in eyes up and right and eyes up and left. In eyes down and right the left hyperphoria increases to 20 △ and is 5 △ in eyes down and left. Hence, there is a paralysis of the left superior oblique. This card is kept as permanent record.

also be brought out on the tangent curtain by placing the red glass first before the paretic eye, then before the sound eye.

When the tests are made in the primary position only, as is so commonly done, it is often overlooked that a hyperphoria in this position may be a hypertropia in one field and not even a hyperphoria in some other field, and other cases showing so little hyperphoria in the eyes front position that they are considered normal may by increasing in a certain field be the whole cause of the patient's symptoms.

When examining in the primary position for distance or at the near point with either screen and parallax or Maddox rod, the tests should be made both with and without glasses if the vision will permit, as a poorly centered glass may produce a false hyperphoria.

A head tilt is a common finding in hyperphoria and affords some interesting study. A hypertropia with the head held straight is often only a hyperphoria with the head tilted. It is evident that in many cases this is the reason for the tilting of the head. On the other hand, the head may be tilted in exactly the same way when it is definitely proved that fusion is not present and when suppression is complete. One case was seen in which, due to injury, the right eye had a complete

optic atrophy. The vision in the left eye was normal, but there was complete paralysis of the external rectus muscle. The head was turned well to the left, and the secondary deviation of the blind eye was so marked that the eye was constantly at the inner canthus. In this case and in total suppression, a sense of muscle imbalance must be the explanation of the marked secondary deviation and head tilt.

The statement is often made that the head is tilted toward the normal field of action of the paretic muscle. This is so regarding the lateral rotators. It is true but part of the time when speaking of the elevators and depressors.

An unpublished article written by Alexander Duane touched on this, and in another he demonstrated why this is so. His final conclusions were that when the head is tilted to obtain fusion, it is toward the higher images instead of always into the field of the paretic muscle. Thus with a paralysis of a right superior rectis, the image of this eye is the higher. If, then, there is an homonymous

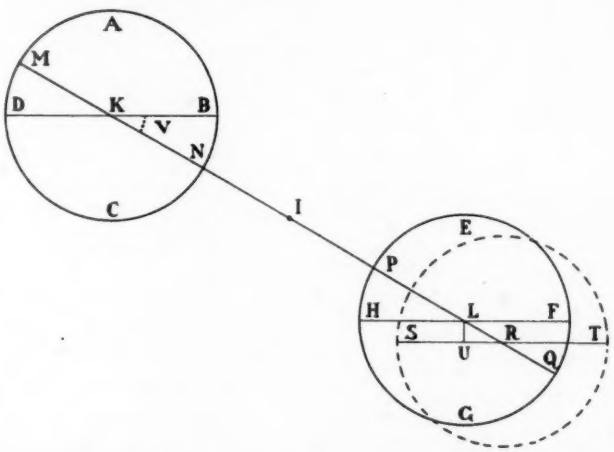

Fig. 2.—*ABCD* is the retina of the left eye, and *EFGH* that of the right eye, each from behind. The right eye is tilted down and the left eye up through the angle *BKL V*, round the median point *I* of the intercentral line. (*I* is here represented as projected on the plane of the retina.) The equator *DB* of the left eye and the equator *HF* of the right eye are supposed to remain horizontal in spite of the tipping. (Actually they tilt with the head, so as to occupy the positions *MN* and *PQ*.) Both eyes are fixing the same object whose image, therefore, falls on the fovea, *K* and *L*, of the left and right eye. There is then single vision. Suppose now that the right eye converges so as to throw the fovea out to *R*. The equator, still remaining horizontal, will now occupy the position *ST*. The retinal image of the object looked at will still occupy the point *L* from which the fovea has receded. *L* is now above the equator by the distance *UL* and to the left of the fovea by the distance *UR*. It will, therefore, be projected to the right of the object of fixation and beneath it. Accordingly, instead of one object there will now appear two: one, *O*, seen by the fixing eye, and the other, *O'*, to the right and lower, seen by the deviating eye. It is easy to see from the construction and to prove mathematically that the more *V*, the angle of tilting, increases, the higher *L* will rise above *R* and the lower *O'* consequently will appear to sink. Hence, the more the head is tipped to the right, the more the right-hand image drops. In this case the right-hand image belongs to the right eye. This figure and also figure 3 were drawn by Dr. Duane and are printed by permission.

diplopia associated with the vertical diplopia, the head will be tilted to the right. However, when there is a crossed diplopia present the head is tilted to the left. This I have observed many times (figs. 2 and 3).

As a general rule, when an elevator is paretic, the head is tilted back, and, when a depressor is involved, the tilt is forward. In this the general rule is followed, i. e., to tilt the head toward the paretic muscle. Not infrequently the head is tilted so as to increase the separation of the images and so make suppression easier. This was observed in a juggler who had paresis of the superior oblique muscle. He could continue his work when the separation of the images was wide enough to make the false image so faint there was no chance of mistaking it for the true image.

When examining a case of hyperphoria, the head must be held straight for the first tests both for distance and near and in the upper and lower corners. However, an interesting and often helpful finding is next to measure the deviation with

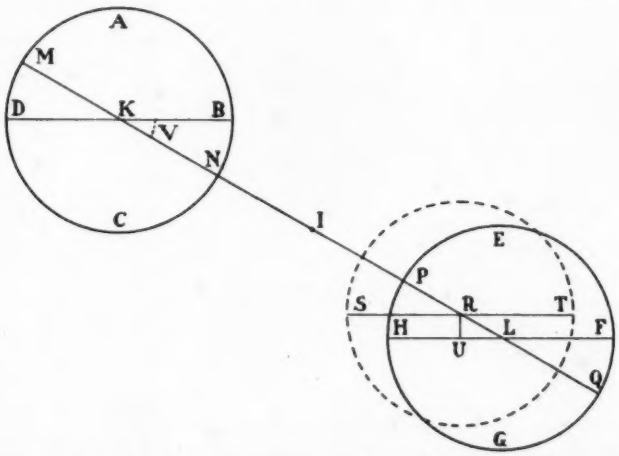

Fig. 3.—This figure shows what happens when with the head tilted to the right shoulder the right eye diverges. In this case the retinal equator of the right eye is carried above the retinal image *L* (from *HF* to *ST*) and inward, so that *L* being below the equator and to the right of the fovea is projected to the left of the object of fixation and above it. Instead of one object, then, there will appear two: one *O* seen with the fixing eye, the other *O'*, to the left and higher, seen with the deviating eye. The more the head is tilted, the more *O'* will appear to rise. Here, again, it is the right-hand image *O* that gets lower as the head is tipped over to the right, only now this image belongs to the left eye.

Similar diagrams may be constructed for all the various deflections of the eyes, vertical, lateral, and compound.

the head tilted in its usual position and then exaggerated by tilting the head to the opposite field. In mapping diplopia on the tangent curtain, the head should likewise be held straight for the first test, but at times additional information is to be had by allowing the head to be tilted into the chosen position and then exaggerated by tilting to the other side.

The treatment of hyperphoria varies as the ophthalmologist's experience increases. Often the symptoms are due to ametropia, which, when properly corrected, relieves the symptoms, hyperphoria being overcome by the power of sursumvergence, by head tilt or otherwise.

The rule to correct two thirds of the hyperphoria by prism has so many exceptions that it is unsafe to follow. A great percentage of ordinary vision is in eyes moderately down; for this reason, if the hyperphoria is due to a paresis of an elevator, the correction by prism may be two-thirds or even less; however, if the hyperphoria is due to a paretic depressor, a full correction is often required, and, at times, even more to correct the increasing hyperphoria in looking down. In low amounts the prism is usually added equally to each lens; in the higher amounts, however, a better correction is often made by putting more prism on the sound eye to correct better the secondary deviation. This applies also to lateral correction by prism when due to a paretic externus or internus.

In correcting a simple hyperphoria or a double hyperphoria in which one predominates over the other, the test is usually made in the position of eyes moderately down. The light is placed from 30 to 45° down at about 3 feet distance. The distance below the eyes front position varies as that particular person's use of his eyes. For a person who uses his eyes more in the upper field, the test is made with eyes moderately up.

On the results of these tests, I prescribe the prism I think to be best for the case in hand. The more noncomitant hyperphorias, either single or double, usually require special consideration. Here a portion of one or both lenses may be frosted. Prism pasters are sometimes used with good results. In those patients who cannot be made comfortable in any other way, the whole lens may be frosted or the association of the eyes otherwise discouraged.

A few cases of paretic muscles have seemed to be benefited by prism exercises. Used for this purpose, the sound eye is held tightly closed by the fingers, and a 20 △ or 30 △ prism is put on and off before the affected eye, the apex of the prism being placed toward the paretic muscle. This in selected cases is worth a trial.

The rotations into the different fields, I believe, is unwise in muscle paralysis, as the secondary deviation is increased rather than the paretic muscle benefited.

Any systemic condition, whether acute or chronic, should be thoroughly investigated and treated. Intestinal toxemia and ptomaine poisoning probably cause more transient hyperphoria than any other condition.

OPERATIVE PROCEDURES IN PHORIAS

Operative procedure should not be resorted to until other means of relief have been thoroughly tried, such as treatment of constitutional causes, prism for wear or for exercises and the various other methods of relief. No operation should be undertaken unless there is a reasonable chance for improvement of symptoms.

Many operative cases are not relieved, not because an operation was contraindicated or was improperly performed, but because the wrong muscle or muscles were chosen on which to operate.

As a general rule, in hyperphoria a resection or tucking of the paretic muscle is preferred over an advancement, first, because of the relatively small amount of correction desired and secondly, because the old insertion is used and a torsion is less liable to result. In some cases tenotomy of the associate antagonist is a better procedure. An example of this is tenotomy of an inferior oblique muscle for paresis of the superior rectus of the opposite eye. The lowest amount of hyperphoria corrected by me in this way was 5 Δ in the primary position, which increased

rapidly in looking up into the field of the paretic muscle. In some of these cases, however, a resection of the superior rectus is preferable; for example, when the paresis is not marked and the secondary deviation is slight.

In paralysis of a superior oblique muscle, the operation of choice is tenotomy or recession of the inferior rectus of the other eye. I have done this and have advised it in several cases with uniformly good results. Here again operation was done on the associate antagonist.

Cases in which the wrong muscle is chosen for operation are commonly seen. Given a case, for example, of right hyperphoria. As the right eye is the higher, tenotomy of the right superior rectus is done. But the right hyperphoria is due to a paretic right inferior rectus, and following the operation there is a paretic superior rectus of the same eye added to the previous condition.

Cases have been observed in which the left superior rectus was advanced to bring the left eye up to the right, where the original trouble was a paretic right inferior rectus. The way to ward against this is by the careful measurement in the upper and lower corners, the only way to be sure of the correct diagnosis.

CONCLUSION

In conclusion, may I emphasize the following:

1. Most cases of hyperphoria are due to a paretic elevator or depressor.

2. In examining every case of hyperphoria the amount should be measured for distance and near, but more important than this, it should be measured in the six cardinal fields, especially the upper and lower corners.

3. The screen and parallax test with the amount of deviation measured by prism is the test best adapted for the corner.

4. The deviation for distance and near may be made by any of the multitude of tests, but, since the screen must be used in every case, it is more convenient to use this method for the primary position also. In this position the screen-Maddox rod, Maddox rod alone, phorometer or any favorite test may be used when desired.

5. When the diagnosis has been made, determine the etiology and institute treatment systemic or otherwise, as indicated.

(*a*) In prescribing prisms for wear, each case must be considered as to amount of hyperphoria, the field in which the hyperphoria increases and whether prism can be used for the correction of this or not. These tests should be made with prisms in the trial frame, and the usual habits simulated as closely as possible.

(*b*) Always keep in mind that some patients with hyperphoria have learned so well how to overcome it, that it may not be the cause of symptoms and should be disregarded. Often the only way to decide this is to prescribe prisms with the proviso that they are to be taken off if not well borne.

(*c*) Frosting some portion of one or both lenses often gives marked relief. To experiment with this, in the office, cover the desired area with adhesive. This often gives a very definite idea as to the wisdom of frosting, and if frosted, how much.

In those cases in which relief is not to be had in any other way, it may be best to frost the whole lens or in some other way dissociate the eyes.

6. Contraindications for operation are:

(*a*) When there is still a possibility of a change in the amount or character of the hyperphoria. This may be due to a change in the course of the disease or as a result of treatment, systemic or otherwise.

(*b*) When the patient has not suppression of the false image, but because it is so far from the true image, it does not annoy. When an operation is performed in this case, the false image may approximate the true image so closely that it would be very troublesome, if for any reason the patient was unable to fuse the images.

7. When operation is decided on, determine whether to (*a*) resect the paretic muscle, (*b*) tenotomize the associate antagonist or (*c*) tenotomize the direct antagonist. The choice will depend on the amount of paralysis and also on the amount of the secondary deviation and the secondary contracture. Not infrequently, the operator may have to resort to any two or all of these procedures.

Most important of all is to study well the corners in all cases of hyperphoria.

EXTENSIVE IRIDODIALYSIS; OPERATION, REATTACHMENT

A REPORT OF TWO CASES

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Minor degrees of iridodialysis are frequently observed, and are known to be corrected in many instances by the simple measure of instillation of atropine immediately following the injury. Many of these, when atropine fails to correct them, cause no visual disturbance and may be insignificant in appearance. But extensive defects of this nature, involving one-fourth and more of the basal attachment of the iris, are relatively rare in cases in which the integrity of the globe is retained and the question of visual efficiency is at stake. These cases present a very striking deformity when viewed in any detail, which is often of as much concern to the patient as is the visual disturbance. The correction of this extensive defect has been reported also to have been accomplished by the use of instillation of atropine. But when this emergency treatment fails, if the globe is retained, and the symptoms justify interference, the following case reports offer a simple operative procedure that may be employed for its correction.

REPORT OF CASES

CASE 1.—A boy, H. D., aged 16 years, who consulted me on Oct. 8, 1930, one year previous to this date, had been struck in the left eye by a golf ball. He complained of no pain or inflammation of the eye, but that the vision had been lost, and that the "pupil," as he expressed it, was white and distorted in outline.

On examination, it was found that the eye was entirely quiet. The cornea was clear. The anterior chamber was deep. The upper temporal one fourth of the iris had been torn from its base, and lay in folds drawn back against the capsule of the lens, but in other respects the iris appeared to be normal (fig. 1 *A*). The pupil was correspondingly displaced and somewhat oval. The lens was opaque and had undergone partial absorption so that now it was about one-third its normal thickness. Under the slit-lamp the capsule of the lens appeared to be intact, and the torn margin of the iris showed a few whitish-gray strands, evidence of atrophic and fibrous change. The tension of the globe was normal. With the ophthalmoscope, only the faintest retinal reflex could be detected in the extreme periphery of the area exposed by the iridodialysis. Light projection was sensitive, and hand movements could be detected. The right eye was normal in every respect.

Submitted for publication, Dec. 10, 1931.

Read at Meeting of Section of Ophthalmology, New York Academy of Medicine, Nov. 16, 1931.

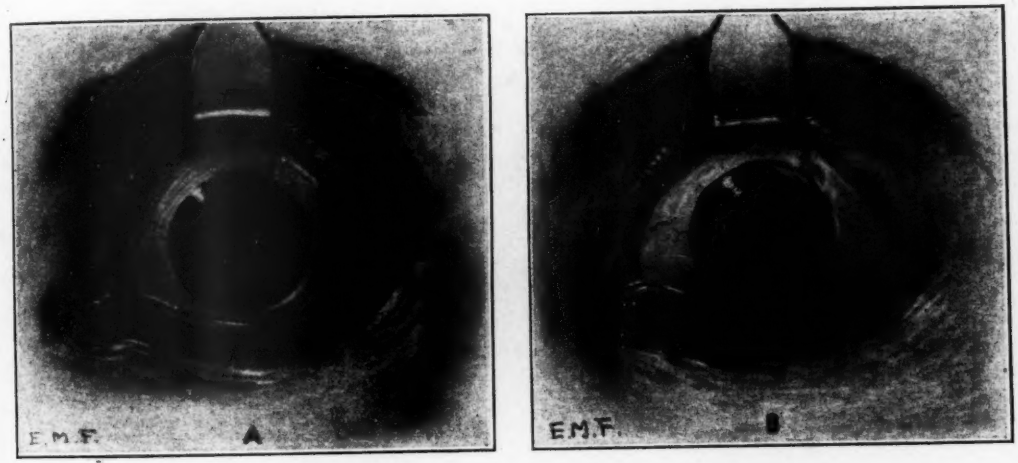

Fig. 1.—*A*, the eye in case 1; *B*, the eye one year after operation.

Fig. 3.—*A*, the eye in case 2; *B*, the eye two months after operation.

A definite method of procedure was decided on, i. e., first to perform the Ziegler discission of the substance and capsule of the lens, carrying the incision into the vitreous, and immediately thereafter to replace the detached iris by means of the Ziegler knife-needle, or if this failed, by means of sewing the torn margin of the iris in the iritic angle with a fine silk (French silk no. 000) suture.

This procedure was carried out in the following manner: With the pupil under moderate dilatation, moderate because the upper temporal one fourth of the iris did not respond to atropinization, the substance and capsule of the lens were

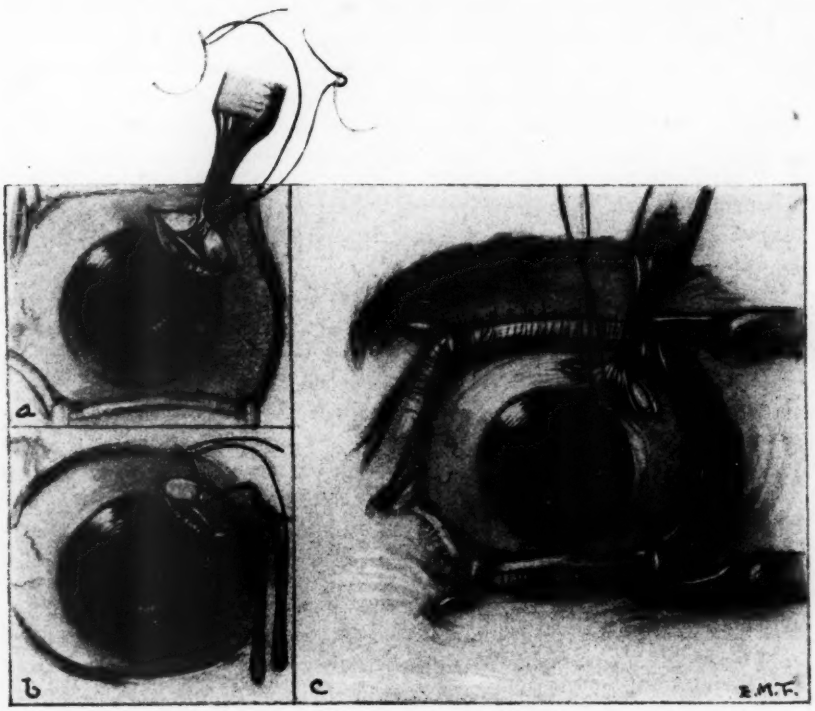

Fig. 2.—Operative procedure in extensive iridodialysis.

divided after Ziegler's method. The point of the knife-needle was then directed against the torn margin of the iris in an attempt to pin these fibers back in the extreme peripheral portion of the iritic angle, but cautiously against corneal substance. With each attempt the torn iris would spring back as before without being caught in the desired position. This method being of no avail a fairly broad keratome incision (about 4 mm.) was made directly at the location of the iridodialysis and in such a manner as to secure a small bit of sclerotic with the large conjunctival flap, similar to that which is desirable in making the modified LaGrange operation. The torn margin of the iris was now grasped with forceps and the iris slightly withdrawn when a fine silk suture (no. 000 French silk) was placed through it by one turn of the needle (fig. 2 *a*), and the iris permitted to spring back as before. This double-armed suture was then carried through the small scleroconjunctival flap at the limbus (fig. 2 *b* and *c*), and the amount of traction on this, when tied, brought the iris into the desired position. Two sutures were then placed in the conjunctival flap under a bit of tension and for the

purpose of support. A solution of atropine, 1 per cent, was instilled and a dressing applied to both eyes. Whether or not the subsequent swelling of the substance of the lens, pressing against the sphincter of the iris, was an aid in supporting it, thus lessening the tension on the suture attachment, is uncertain, but for this reason it seemed desirable that the two operations be performed at the same time. A firm cotton dressing was applied every second day for two weeks. The suture in the iris was removed on the fourth day.

The result as seen in figure *B* (now more than a year after operation), is a satisfactory reattachment of the base of the iris, with slight irregularity of the surface of the iris; the pupillary margin is free, and there is no visible area of iridodialysis at either side of the reattachment. The pupil is round above, with only the slightest depression below, which was due to an old minute attachment to the capsule of the lens. The pupil, furthermore, is equal in size to that of the fellow-eye, and is about equally sensitive to light and accommodation. The substance of the lens has completely absorbed, and the pupillary area is clear. There is a bit of pigment at the point where the suture was brought through the scleroconjunctival flap, but there is no prolapse of the iris nor has there been any prolonged iritic reaction or pain or headache. With a correcting lens the vision of the eye is 20/16.

Case 2.—A boy, W. H., aged 13 years, was referred to me by Dr. Henry G. Wincor, at the New York Eye and Ear Infirmary, on Sept. 29, 1931. On Jan. 1, 1929, two years and nine months before, he had been struck in the left eye by a BB shot, when almost immediately there was total loss of vision of the eye. He had been admitted to the Fordham Hospital, where he was confined for two weeks. He stated that the treatment there consisted of hot fomentations and atropine, and that a roentgenogram at that time had revealed no evidence of a foreign body. He complained of no symptoms other than the loss of vision, when he was admitted to the Infirmary on Sept. 29, 1931.

On examination, it was noted that the eye was quiet, the cornea clear, the anterior chamber deep, the lower nasal one fourth of the iris had been torn from its base and lay in folds, drawn back, but free of any attachment to the capsule of the lens (fig. 3 *A*). In other respects the iris appeared to be normal. The pupil was displaced and somewhat oval in the meridian of 135 degrees. The lens had been completely absorbed, and now a fairly thick secondary membrane remained. Under the slit-lamp the capsule of the lens was noted to be folded back evenly beneath the iris, but the posterior membrane remained intact, no vitreous hernia being present. Along the torn margin of the iris there could be noted a few whitish-gray strands, evidence of atrophic and fibrous change. The tension of the globe was normal. With the ophthalmoscope no retinal reflex could be detected in the area exposed by the iridodialysis. Light projection was sensitive, and hand movements could be detected. The right eye was normal in every respect.

In the treatment of this case, a similar method of procedure was followed as that employed in case 1, except that reattachment of the iris was performed without discission of the membranous cataract. But the simple technic of reattachment by means of a suture through the scleroconjunctival flap was in every respect the same as that employed in case 1.

The eye was dressed on the second day after operation, when it was noted that the iris was reattached and without folds or irregularity in its surface; the pupil was partly dilated but round and regular. The injection of the eye was surprisingly moderate in extent and character. On the fourth day, the sutures were removed. Two days later, it was noted that there had occurred a slight retraction of the

sphincter of the iris at the angle of reattachment, and the wound was slightly protruding or edematous as though some filtration of aqueous had occurred after removal of the sutures. Furthermore, the obstreperous activity of this youth in the hospital at that time may have been a contributing factor in causing this evident escape of aqueous through the wound. He was returned to his bed after a firm cotton dressing had been applied, and the wound healed promptly.

It is seen in figure 3 *B* (now two months after operation) that the level of the iris is drawn very slightly forward in its reattached portion; there are no folds or unattached lateral portions; the pupil is oval in the meridian of 60 degrees, with retraction of the sphincter of the iris to about one-half the width of the iris (1.5 mm.). The upper pupillary margin is round and regular, and there are no posterior synechiae. The reaction to light is normally sensitive, except in the small retracted portion where a sluggish response is noted. There is no visible pigment in the iris, beneath the scleroconjunctival flap, nor is there any prolapse of the iris. There has been no prolonged iritic injection, nor has the patient complained of pain in the eye or headache.

COMMENT

In the surgical treatment for this traumatic defect, it is obvious that every consideration must be given the possibility of doing more harm than good. In other words, do the end-results justify the means and risk of operation? The extent of the iridodialysis in the individual case, and therefore the deformity and the possible visual disturbance, may be offered by some as sufficient justification for it. It is certainly true that the visual disturbance in most cases is limited; in many cases it causes no visual disturbance whatever, but in other cases the diffusion of light is continually confusing, especially for field vision. But if the defect is present in the only eye or in the "master" eye of the patient, of course the visual disturbance becomes accordingly more acute, and therefore its correction becomes essential to his visual efficiency. The deformity occasioned by this defect varies with its location and the prominence of the eye. While the operative correction of it for this purpose alone should remain entirely within the choice of the patient, it is not surprising that often this is the principal reason for having it corrected, when the defect is located in the lower portion of the eye, and when viewed in any detail. I do not advocate this operative procedure merely for the purpose of correcting a defect that may be corrected, as is sometimes the urge to surgeons, who may be inclined to attempt to "paint the lily white." The indications here are clearly understood. The patient should be advised accordingly.

This leads at once to the means and the risk of the procedure. Except for the hazard of infection and the delicacy of the very simple technic, the operative risk cannot be regarded as very serious but may be classed with that encountered in the average preliminary iridectomy. If the iris fails to be incarcerated, the same defect remains; if the iris is drawn out a little too far, the defect is improved and the pathologic

process in the defect remains the same. Prolapse of the iris obviously must be avoided, but the substance of the iris will not be torn or destroyed by delicate and cautious manipulation in this operation.

As to the operative technic, three important points of advantage may be emphasized. These are: (1) the location and breadth of the keratome incision; (2) the necessity of catching the very margin of the torn iris by the suture, and (3) the traction on the iris, when the suture is tied, permitting the operator to judge the amount of incarceration that is necessary to correct the defect. The keratome incision, made in the iritic angle and with a small scleral flap should be about 4 mm. in width (not more than 5 mm.). The ease with which incarceration of the few fibers of the iris takes place, with gentle traction on the suture, impresses one with the importance of a small incision, and yet it must be large enough to permit a few lateral fibers of the torn iris to engage it. Too much importance cannot be placed on the necessity for passing the needle through the very margin of the torn iris. This is true not only for very obvious reasons in correcting the defect, but also because this torn margin is partly fibrous and therefore holds the suture quite securely for the purpose of traction. The matter of traction is interesting as well as important in this connection, because after a year's time the torn section of the iris behaves like a bit of firm rubber, and when drawn through the wound with forceps, if released, snaps back in its original position. This was particularly true in case 1, but to a less degree in case 2. On the other hand, only a few fibers of the iris are necessary for securing satisfactory incarceration, and the tension required is adjusted by the suture when it is tied. This tension on the section of the iris should be slightly in excess of that which permits the pupil to appear round at the time of operation.

Since traumatic cataract or membranous cataract is frequently associated with extensive iridodialysis, the question of procedure in this event is worthy of consideration. Especially is this true because it may be found to be not only more expedient but also an aid in securing a more perfect pupillary correction, if the anterior chamber is maintained following discission of the capsule and the presence of vitreous in the chamber thus supports the pupillary margin of the iris. This very reasonable assumption is demonstrated by the satisfactory result of this procedure in the first case here reported. Furthermore, the hazard of two operations is limited because of the combined procedure. It must be admitted, however, that in the second case the retraction of the iris beyond the normal position could not be attributed to lack of support from the pupillary margin but was due to filtration of aqueous following the removal of the suture and perhaps the unruly disposition of the patient at that time. Although the anterior chamber was collapsed

during the procedure in this (second) case, the technic was carried out with the same apparent degree of certainty as to incarceration as in the first case. But the question of support of the anterior chamber by the pressure of the vitreous through the pupillary area, thus limiting the tendency for the anterior chamber to collapse and so permitting excessive incarceration in the filtering wound, must be considered of some importance when one analyzes the forces affecting the immediate and subsequent position of the reattached iris.

Regarding the postoperative care and treatment in these cases, suffice it to say that atropinization is not so necessary as one may at first believe it to be. The moderate iritic reaction following this operation may be attributed to the pathologic changes in the section of the iris after its long inactivity, as well as to the absence of undue traction on the iris and ciliary body. At the second dressing, (the eye being dressed every second day), pilocarpine may be instilled and without noticeable iritic reaction. I believe it is essential, however, that the patient remain in bed and under quiet conditions for forty-eight hours after the suture is removed on the fourth day. For this reason it seems inadvisable to operate on children at an age when their full cooperation in the postoperative care cannot be depended on. Both eyes are bandaged for the first forty-eight hours after operation.

The pathologic changes in simple extensive iridodialysis uncomplicated by cyclitis, hypertension, penetrating wound, dislocated lens, atrophy of the iris or destructive tears of the iris, is seldom observed under the microscope, because these eyes are usually retained. In complicated cases, atrophy or necrosis of the substance of the iris takes place as the result of a nutritional disturbance due to the altered blood supply through trauma, hypertension or the organization of exudate. It is reasonable, therefore, to assume that in uncomplicated iridodialysis the pathologic process is limited to the changes along the torn section of the iris, where fine fibrous bands appear and a minor degree of atrophy is present because the blood supply is relatively undisturbed. This may account for the firm rubber-like consistency of the section when withdrawn during operation for inserting the suture. This also, no doubt, permits the suture, when traction is made, to hold firmly a few fibers in the lips of the keratome incision, as it draws the section of the iris laterally, and thus separates its folds.

It is of importance to note that no part of the suture remains in the anterior chamber of the eye, but with traction its presence in the wound no doubt aids in the production of fibrinous exudate which fastens the lateral portions of the torn margin of the iris along the inner lips of the wound. The importance of using a suture instead of causing simple incarceration of the iris in the wound is borne out by theory, by actual

manipulation at the time of operation, by the pathologic process in the part and the forces that act in controlling the position of the iritic section, as well as by the results reported. That the suture or the fibers of the iris, or both, may act as a temporary seton, and thus delay early healing of the wound is an important consideration in the postoperative care, since excessive retraction of the section of the iris may result. It seems reasonable, however, that excessive retraction of the iris may also be due to a badly injured iris section which reacts thus after unfolding from traction by the suture, and which obviously cannot be avoided.

The application of this method of operation in minor degrees of iridodialysis may be considered advisable only when the symptoms may justify it. The technic would be relatively simplified and the result therefore relatively more certain. I have observed but one case in which it seemed advisable. This was one in which a small (4 mm.) but abrupt iridodialysis in the nasal angle of the right eye caused constant confusion of images, especially for near objects and when reading. Operation was not urged, but the possibilities were explained in some detail.

REVIEW OF LITERATURE

Although numerous cases of iridodialysis have been reported with special reference to treatment and results, there are only a few reports in which operative treatment has been attempted, and in these many details are not recorded and no precise drawings of the defects and the results of operation are presented.

Cases of spontaneous reattachment of fairly large defects in the iris have been reported in recent years by Shedlow,[1] in 1924, by Brown,[2] in 1924, and by Duane,[3] in 1926. Duane attributed the spontaneous cure in his case to repeated hemorrhages forming a fibrinous exudate, the continuous traction of which gradually drew down the detached iris and glued it into place.

In their textbooks, de Schweinitz, Fuchs and Römer suggest no operative measures for the correction of this defect. From this one may judge that either the defect does not need correction as a rule, or the operative procedure is not justified by the risk entailed.

The operative treatment dates back to the operation of Critchett [4] in 1892, who revised the operation of Adams, iridencleisis, a procedure for

1. Shedlow, Abraham: Traumatic Iridodialysis with Complete Reattachment of the Iris, J. A. M. A. **83**:1507 (Nov. 8) 1924.

2. Brown, J. C.: Reattachment of the Ruptured Iris, J. A. M. A. **83**:1865 (Dec. 6) 1924.

3. Duane, A.: Am. J. Ophth. **9**:531, 1926.

4. Critchett: Tr. Soc. United Kingdom, 1892.

making an artificial pupil by displacement of the iris in a keratome incision. Although this operation was not performed by Critchett for iridodialysis, the principle of incarceration of the iris was the same. Smith,[5] in 1891, reported four cases in which his modification of Critchett's operation of iridodesis was done, and the smallest possible portion of iris was drawn into a small peripheral wound. In his report no details are given as to the character of the iridodialysis or the results secured. Alt,[6] in 1916, claimed to have used this technic in the surgical treatment for iridodialysis, but no case reports were recorded.

In 1909, Jameson [7] reported four cases and the results of operation by an ingenious method of suturing the torn margin of the iris, after inserting two needles into the anterior chamber in order to catch the iris section in the sutures. The purpose of this method was to bring about a union of the torn margin of the iris with a freshened surface within the anterior chamber without incarceration in the corneal wound. Although theoretically applicable in the correction of the defect, the method presents certain complications which Jameson pointed out.

In 1920, Bulson [8] reported briefly three cases of iridodialysis in which he believed operation was justified. In one of these a suture was used through a keratome incision, and the result was that "the pupil was drawn upward to a considerable extent, yet the result was satisfactory." In the second case simple incarceration (without a suture) in the corneal wound was attempted, but the iris fell back in the anterior chamber. In the third case, a slight prolapse of the iris in a small wound was purposely attempted (without a suture), and tincture of iodine was applied to the wound. Althought there was irregularity of the pupil, the result was regarded as very satisfactory.

CONCLUSIONS

1. The correction of iridodialysis by operative procedure offers an interesting problem in the analysis of the forces affecting incarceration of the iris for this purpose.

2. The first case demonstrates the degree of correction that may be secured by this simple operative technic: a pupil perfectly round and equal in size to that of the fellow-eye, an active sphincter irides, the base of the iris apparently reattached, the level of the iris almost normally maintained—in fact, no deformity and the visual efficiency restored.

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5. Smith, Eugene: Tr. Ophth. Sec., A. M. A., 1891, p. 285.

6. Alt, A., in American Encyclopedia and Dictionary of Ophthalmology, edited by Casey A. Wood, Chicago, Cleveland Press, 1916, vol. 9, p. 6594.

7. Jameson: Arch. Ophth. **38**:391, 1904.

8. Bulson, A. E., Jr.: Am. J. Ophth. **3**:357, 1920.

3. The second case demonstrates the result of fibrous contraction in the substance of the iris or of excessive incarceration of the iris following filtration of aqueous, which occurred after removal of the suture on the fourth day. Prior to this, the correction of the defect was in every respect as satisfactory as that in the first case. The pupil is displaced, but the deformity is improved and the vision may be restored following discission of the secondary membrane.

4. While it is quite obvious that this simple operative procedure requires some delicacy in technic, which must be emphasized, it is neither difficult nor uncertain in its performance.

5. The hazard of operation is in direct proportion to the extent and location of the defect, and should be measured in terms of the deformity and the probable improvement in the visual efficiency.

ISOLATED FOCI OF CALCIFICATION IN THE SCLERA

ANATOMIC AND CLINICAL ASPECTS

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There are but few reports in the literature of localized calcareous degeneration of the sclera. The earliest of these was referred to by Pagenstecher [1] as having appeared in the *Blasii obs. med. ra Amstel.* of 1677, concerning a case of circumscribed "ossification" of the sclera, which he believes to have been only a calcification, since osteoid transformation of the connective tissue does not seem to develop in the sclera.

In 1860, Pagenstecher [1] himself reported a case of isolated calcification of the sclera, anterior to the insertion of a lateral rectus, in an atrophic eye. After extraction of the lime salts, the scleral fibers in the affected region were of normal appearance.

Katz [2] was the first to point out the possibility of a primary localized necrosis of the sclera and a secondary deposition of calcium salts, as in his case the scleral fibers, after extraction of the calcium salts, were seen to be greatly changed, and this region, as well as the surrounding sclera, was devoid of nuclei. The calcified area in this case was located just anterior to the external rectus insertion.

This report adds to the limited group of known cases of this regressive metamorphosis an anatomic study of two eyes and also three clinical cases in which calcification of the sclera was encountered surgically.

ANATOMIC STUDY OF TWO CASES

CASE 1.—This histologically studied bulb (received from an outside hospital by the Ophthalmic Laboratory of Rush Medical College) was the left eye of an aged woman of 106 years, which had been totally blind from secondary glaucoma for four months prior to enucleation. The rather scanty clinical data available indicated that there had been recurrent attacks of painful inflammation and failing vision of this eye for about five years before it was excised, at which time the

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Submitted for publication, Dec. 9, 1931.

From the Department of Ophthalmology, Rush Medical College of the University of Chicago.

1. Pagenstecher, A.: Beiträge zur pathologischen Anatomie des Auges, Arch. f. Ophth. **7**:118, 1860.

2. Katz, D.: A Localized Area of Calcareous Degeneration in the Sclera, Arch. Ophth. **2**:30 (July) 1929.

intra-ocular tension was elevated to 90 mm. of mercury, the anterior ciliary vessels were deeply engorged, the cornea was dull and clouded, there was an iris bombé with an extremely shallow anterior chamber, and the lens was totally cataractous. The enucleated bulb, which was of normal size (24.0 by 23.5 by 23.0 mm.), was fixed in Zenker's fluid and embedded in celloidin. The eye was sectioned serially, every tenth section was stained with hematoxylin-eosin, and special chemical and staining reactions of additional sections were studied as hereinafter described.

Histologic Summary.—The corneal epithelium was hydropic and rather irregular. There was pannus degenerativus, superficial to Bowman's membrane, which was intact. The corneal stroma was of normal appearance. The anterior chamber was very shallow, and was partly filled with blood. There were extensive anterior synechiae of the root of the iris, which was atrophic, and its pupillary margin was fragmented and deeply embedded within a thick cicatricial membrane spanning the pupil. This granulation tissue contained numerous foci of leukocytes and curl-

Fig. 1 (case 1).—Focus of calcification in the sclera, stained with hematoxylin-eosin.

ing fragments of lens capsule. There were calcium deposits beneath the capsule of the lens. Clumps of polymorphonuclear leukocytes were present in the posterior chamber and also in the vitreous body, especially as precipitates on the retina. There were much leukocytic infiltration of the inner layers of the retina and a moderate periphlebitis, without other pathologic changes. There were no visible pathologic changes in the choroid, which was thinned (shrunken) by fixation. There was a deep ampulliform (glaucomatous) excavation of the optic papilla. The sclera was rather poor in nuclei throughout, and appeared thinner (fig. 1) than normal near the equator, where it was reduced to a thickness of 0.40 to 0.45 mm. Just anterior to the insertion of the internal rectus muscle there was an area, entirely devoid of nuclei, within which there were heavy, dark blue-stained calcium deposits. This calcified center measured 3 mm. horizontally, 6.5 mm. vertically and 0.25 mm. in width. Its central portions were almost structureless, with only a scattered faint striation, and the marginal regions were composed of innumerable fine, blue-stained droplets, gradually decreasing in number toward and fading into the contiguous sclera.

CASE 2.—This histologically studied bulb was the right eye of a man of 84 years, in which there was a severe postoperative inflammation with a ring abscess of the cornea, following an unsuccessful attempt to remove the lens, during which the lens was dislocated backward into the vitreous. The eye was excised about fifteen weeks after the attempted lens extraction, and was sent in from an outside hospital to the Ophthalmic Laboratory of Rush Medical College for histologic examination. The bulb, which measured 24 by 23 by 22 mm., was fixed in Zenker's fluid, embedded in celloidin and sectioned and stained as in case 1.

Histologic Summary.—The corneal stroma was richly vascularized near the limbus by superficial and deep vessels, and there was moderate round cell infiltration between the lamellae. The luxated lens nucleus in the vitreous was surrounded by a thick layer of cells, consisting of polymorphonuclears, phagocytes and giant cells. The shrinking process in the interior of the eye was evidenced by detachment of the ciliary body and choroid, axial detachment of the retina and a bowing inward of the lamina cribrosa. The sclera (fig. 2) was of normal thickness. Just anterior to the insertion of the internal rectus muscle, a slightly blue-

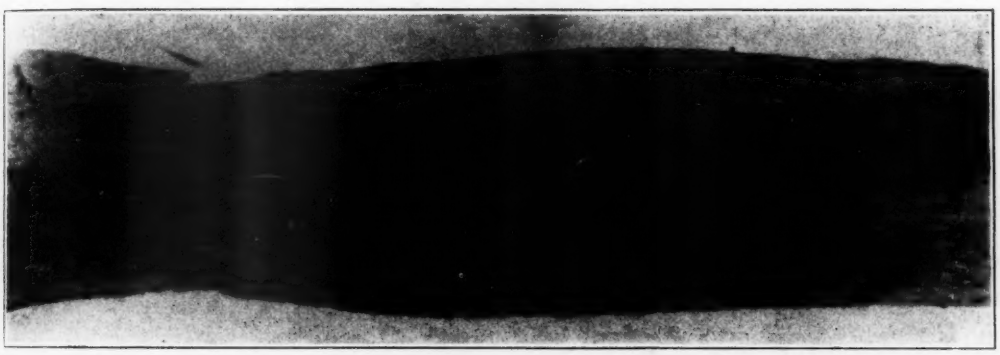

Fig. 2 (case 2).—Focus of calcification in the sclera, stained with hematoxylin-eosin.

stained region of the sclera was devoid of nuclei, and in its central portion there were dense calcium deposits. The calcified area measured 1.5 mm. horizontally, 2.6 mm. vertically and 0.5 mm. in width. The finer histologic structure of this calcified focus was entirely similar to that described in case 1. There was no calcification or ossification of the choroid or of any other tissue of the bulb except the sclera, as noted.

CHEMICAL AND SPECIAL STAINING REACTIONS

On the application of dilute hydrochloric acid to sections of the eye from case 2, there was a free production (profuse generation) of gas bubbles in the calcified portion, as viewed under the microscope. In examining sections of the eye from case 1 by the same method, there was no gas formation except for two or three small bubbles in one section. This chemical reaction indicated the presence of calcium carbonate in considerable amount in the calcified focus in case 2, and a trace of carbonate irregularly distributed through the focus in case 1.

Sections from each eye were also stained with silver nitrate, being immersed for three hours in a 1 per cent solution and then exposed to daylight. The calcified foci in sections from both cases stained quite similarly, i. e., dark brown in the center and light brown in a narrow encircling zone. Kossasch's reaction, the formation of silver phosphate from calcium phosphate and silver nitrate, occurred more rapidly in the area that contained only a low percentage of calcium carbonate, i. e., case 1. This was shown by comparing the density of staining of sections from the two eyes after a shorter period of immersion in the 1 per cent silver nitrate, when there was a darker coloration of the focus from case 1.

Decalcified sections from both cases, some stained with hematoxylin-eosin and others with silver nitrate, exhibited almost identical features. The decalcified area in each case was stained a light tan, being somewhat darker in case 1 than in case 2, the color in the latter being about the same as that of the adjoining sclera. In both cases the scleral fibrils comprising the periphery of the calcified areas were normal in size and contour. Progressing toward the center, however, they became more irregular, and in the central portion they were entirely broken up, leaving a central empty space, with only a few amorphous masses along its wall.

Both decalcified foci stained light purple with hematoxylin-eosin. In the central portions in some sections were several slits with ragged outlines and crumbled remnants of scleral lamellae.

These chemical reactions and special stains demonstrated that in both cases there was not only calcium deposition between the scleral fibrils, but also a degeneration of the fibrils themselves, which must have been progressive, leading to their complete destruction.

The almost identical locations of the calcified areas in the two cases described, as well as in the two cases previously reported by other authors, viz., invariably just anterior to the insertion of a lateral rectus muscle, suggest the idea that mechanical factors create here a locus minoris resistentiae. This explanation is further strengthened by the fact that in my two cases the calcified focus was just anterior to and parallel with the internal rectus insertion. It is well known that the lateral excursions are the eye movements of prevailing frequency, that the interni are relatively more powerful than the externi and that convergence is the predominating movement of the eyes, with the intensive near work that is such an important feature of modern industrial civilization. The close anatomic relation of Tenon's capsule with the muscle on the one hand and with the episclera on the other is a factor that may well produce more mechanical stress and strain on the underlying sclera distal to the muscle insertion than occurs in other regions of the sclera. It may also be possible that old age or chronic diseases have a deleterious

effect even on tissue of such a low metabolic level as the sclera, impairing its physiologic resistance to adverse external influences, so that localized degeneration or necrosis occurs first in these regions.

That these localized calcifications are not devoid of clinical interest is indicated by some surgical experiences of Prof. A. Fuchs[3] several years ago in the first eye clinic of the University of Vienna. He has kindly furnished me for publication in this paper the following clinical data concerning one patient with glaucoma and two patients with cataract.

REPORT OF THREE CLINICAL CASES

CASE 3.—J. R., aged 60, entered the clinic with an absolute glaucoma of the right eye and an acute congestive attack of glaucoma in the left eye. Since the intra-ocular tension was not sufficiently reduced by miotics, although the pupil was narrowed, a von Graefe iridectomy was done on the left eye. As soon as the point of the knife entered the extremely shallow anterior chamber, an unusual resistance was encountered in the sclera, so that considerable force was required to push the knife across the chamber, and in order to make the counter puncture, it became necessary to fix the eye with Elschnig's forceps. During the first part of the section, the movement of the knife through the sclera produced a scratching sound that was distinctly heard by the operator, patient and bystanders. This unusual phenomenon was attributed to a localized calcification of the sclera, which was not subject to anatomic confirmation because the eye fortunately made a good recovery.

CASE 4.—M. G., aged 76, entered the clinic in 1920 for extraction of a senile cataract. Although the knife was tested and found sharp just prior to operation, so much resistance was encountered in the scleral limbus that it was impossible to force the point of the knife into the anterior chamber. The knife was laid aside and a second knife was introduced into the same wound; after much further difficulty, its point entered the anterior chamber, and the knife was pushed across the chamber, making the counter puncture without undue resistance. The sectioning was easy on the nasal side, but there was so much resistance on the temporal side that the knife was turned, first completing the inner and upper part of the section and then, with great difficulty and many sawing movements, the temporal part.

CASE 5.—T. W., aged 62, entered the clinic in 1920 for extraction of a senile cataract. Marked resistance was met in the scleral limbus before the knife entered the anterior chamber. There was continued scleral resistance as the knife traversed the chamber, and similar resistance was encountered in making the counter puncture; the remainder of the section, however, was completed easily, without pathologic resistance. In order to test the sharpness of the knife further, the operator used the same knife for the next patient, and the section was made without any difficulty. Thus it was probable that in the previous case the knife had encountered calcified sclera.

In all three of these cases the resistance was encountered in the lateral portions of the scleral limbus and not in the upper portions, which corresponds well with the lateral location of the calcified areas found in the eyes studied histologically.

3. Fuchs, A.: Personal communication to the author.

SUMMARY

In two histologically studied eyes from very old persons, one removed because of recurrent uveitis and secondary glaucoma, the other because of endophthalmitis and beginning atrophy bulbi, there were localized, well defined foci of calcareous degeneration within portions of the sclera completely devoid of nuclei, indicative of a primary necrosis of the sclera.

The identical location of these calcified areas anterior to the tendinous insertions of the lateral recti in these cases and also in other cases previously reported by other writers indicates that in addition to chronic disease or old age mechanical factors may well be predisposing causes for this localization of the calcified lesions.

The discovery of hard, resistant and apparently calcified scleral tissue adjoining the limbus in the three surgical cases outlined shows that this condition is not without some clinical importance.

DETERMINATION OF THE OXIDATION-REDUCTION MECHANISM IN THE LENS OF RABBITS WITH NAPHTHALENE CATARACT

HAROLD GIFFORD, JR.,. M.D.
OMAHA

The occurrence of cysteine in the normal crystalline lens has been suspected since the work of Arnold [1] in 1910. Arnold considered the positive color reaction obtained with sodium nitroprusside as proof of the presence of cysteine. Jess,[2] in 1922, found that cysteine and its oxidized form, cystine, make up 2.3 per cent of alpha crystallin and 4.9 per cent of beta crystallin, the soluble proteins of the lens, and are absent from the insoluble protein residue. In 1922, von Reis [3] found that the reaction to sodium nitroprusside, which is characteristic of cysteine-containing substances and is positive in normal lenses, was decreased or entirely negative in the cataractous lens. It was absent only in the nuclei of immature senile cataracts, while in mature cataracts it was negative except in the thin layer of cortex next to the capsule. Jess [4] confirmed his findings and obtained similar results in traumatic cataract and cataract produced by feeding naphthalene to rabbits. In the latter condition, lenses with early cataract gave a positive reaction to sodium nitroprusside; those removed three months later showed a negative reaction in a zone around the nucleus, while after six months the reaction was absent except in a zone just under the capsule. In noncataractous human and beef lenses, the reaction was positive as in the normal lens. This indicated that the loss of cysteine did not precede the occurrence of opacities in the lens but accompanied such changes.

A possible significance of the absence of cysteine in the cataractous lens is indicated by the fact that a cysteine-containing substance repre-

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Submitted for publication, Dec. 22, 1931.

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The work was performed with the aid of a grant from the Research Committee of the American Medical Association.

1. Arnold: Ztschr. phys. Chem. **70**:300, 1910.

2. Jess, A.: Arch. f. Ophth. **109**:463, 1922. Jess, A., in Schieck, F., and Brückner, A.: Kurzes Handbuch der Ophthalmologie, Berlin, Julius Springer, 1930, vol. 5, pp. 232 and 266.

3. von Reis: Arch. f. Augenh. **72**:156, 1912; Arch. f. Ophth. **80**:587, 1912.

4. Jess, A.: Arch. f. Augenh. **71**:259, 1912; **72**:156, 1912; Ztschr. f. Biol. **61**:93, 1913.

sents the principal known agent of the oxidation-reduction process that goes on in all living tissue. With its free SH group, cysteine is capable of becoming oxidized to form cystine, which by reabsorption of hydrogen, becomes cysteine again. Von Reis himself suggested that his findings indicated a cessation of metabolism in the cataractous lens. Goldschmidt [5] studied the oxidation-reduction mechanism of the lens, and showed that it consisted of a dialyzable, thermolabile and an insoluble, thermostable constituent. The former he showed was identical with glutathione, which is present in most living tissues, and the latter he thought depended on the presence of cysteine in a form bound to the proteins of the lens. Adams [6] reviewed the work on this subject up to 1925, and added observations of her own. She estimated the oxygen consumption of lenses in a respirometer, and found that this was much increased by the addition of glutathione and was absent after dialysis of the lens in which glutathione is extracted. She estimated the amount of glutathione directly in the normal ox lens and found it to be 0.305 per cent of the whole weight of the lens.

Another method of estimating the oxydation-reduction mechanism of the lens is by the reduction of methylene blue (methylthionine chloride, U. S. P.) by the method of Thunberg. This has been investigated by Ahlgren,[7] Goldschmidt [5] and others. They found that the normal lens reduces methylene blue to colorless form at a fairly constant rate, which Ahlgren ascribed to the presence of a catalase. Goldschmidt found that after extracting the lens with tenth-normal sulphuric acid this reducing power was lost, while the residue still gave a positive reaction to sodium nitroprusside, indicating that cysteine was still present. Adding small amounts of the oxidized form, cystine, restored the reducing power to the residue.

This led Goldschmidt to the aforementioned theory of a soluble and insoluble oxidation-reduction agent. He believed that the insoluble (thermostable) substance (cysteine) served to reduce the glutathione after the latter has been oxidized.

EXPERIMENTAL WORK

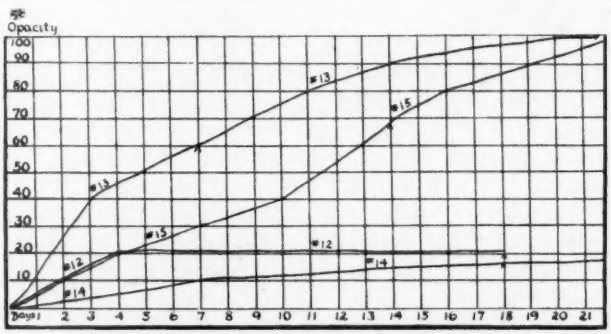
The present study was undertaken to determine the amount of glutathione in the lens of rabbits with naphthalene cataract in various stages, as compared with that in the normal rabbit lens and in a few human lenses with senile cataract. It was also undertaken to study the ability to reduce methylene blue of lenses containing normal and

5. Goldschmidt, M.: Arch. f. Ophth. **88**:405, 1914; **93**:447, 1917; **113**:160, 1924.
6. Adams, D. R.: Brit. J. Ophth. **9**:281 (June) 1925.
7. Ahlgren, G.: Acta ophth. **5**:1, 1927.

reduced amounts of glutathione. The method of determining glutathione employed was the modification of Tunnicliffe's method suggested by Perlzweig and Delrue.[8] Since this work was begun, Adams[9] examined lenses of rabbits with naphthalene cataract and found that no glutathione was present.

Lenses were removed in the capsule from the freshly killed rabbit, weighed and ground in a mortar with 5 cc. of 10 per cent trichloracetic acid for twenty minutes. Thousandth-normal sodium thiosulphate is made up accurately from a tenth-normal thiosulphate solution standardized against a standard dichromate solution and the amount of thiosulphate added that is found necessary by the titration to make the solution exactly thousandth-normal. Thousandth-normal iodine solution is titrated against the accurately titrated thousandth-normal thiosulphate, and a correcting factor is found for change in the concentration of iodine.

The lens extract is filtered into a 20 cc. side-arm test tube through a filter-paper mat which is washed with distilled water to make a total volume of 20 cc. It was necessary in each determination to titrate a similar amount of water containing 5 cc. of 10 per cent trichloracetic acid, since such a blank gave a definitely greater



Course of opacities in a group of animals fed naphthalene. When the cataract had obtained an opacity of 10 per cent, there were water fissures; at 20 per cent, there were wedge-shaped opacities in the cortex; at 40 per cent, diffuse opacity with more radial stripes; at 60 per cent, the red reflex was barely present; at 80 per cent, there was no red reflex but some clear opacities in the cortex and at 100 per cent practically all the cortex was opaque. The arrow indicates when the feeding was stopped. The numbers on the curves indicate the respective rabbits used for experimentation.

liberation of iodine than titrations with water alone, a fact not mentioned by Perlzweig and Delrue. This blank, containing 15 cc. of water, 2 cc. of 25 per cent potassium iodide and 10 cc. of thousandth-normal iodine is allowed to stand for fifteen minutes and titrated against the thousandth-normal thiosulphate solution, from 1 to 2 cc. of 1.25 per cent starch solution being used as an internal indicator. The factor found gives the normality of the iodine solution and is employed in calculating the results of the lens-extract titration, which is carried out in an exactly similar manner.

8. Perlzweig, W. A., and Delrue, G.: Biochem. J. **21**:1416, 1927.

9. Adams, D. R.: Brit. J. Ophth. **14**:49, 1930.

The amount of glutathione is calculated by subtracting the amount of thiosulphate solution used to react with the amount of iodine solution added (corrected for normality). This represents the amount of thousandth-normal iodine neutralized by the glutathione present. Since 1 cc. of thousandth-normal iodine is equivalent to 0.25 mg. of glutathione, the percentage of glutathione is expressed by the foregoing amount times 0.25 divided by the weight of the lens in milligrams multiplied by 100. Thus for a lens weighing 384 mg., the amount of glutathione was found to be 0.133 mg., or 0.347 per cent.

Full-grown rabbits were fed a solution of naphthalene in olive oil by stomach-tube, a dosage of 1 Gm. per kilogram per day being administered until opacities were well developed. Some animals were remarkably refractory to the drug, being fed as long as two and one-half weeks without marked changes developing in the lens, while in others definite opacities were present after from five to seven days, which in some cases went on to complete opacity without further feeding. The course of a group of these animals is shown in the chart.

Twelve human cataractous lenses were examined from cases of mature or nearly mature senile cataract.

A normal rabbit lens was examined in the same way at the time each cataractous rabbit lens was tested.

The reduction of methylene blue was determined with the technic employed by Goldschmidt, with certain modifications. Five cubic centimeters of a buffer solution at p_H 7.8 was used, to which were added 0.1 cc. of 1 : 50,000 of methylene blue and a freshly extracted lens without its capsule. The tubes were kept in a water-bath at 37 C. under a constant negative pressure of 70 mm. of mercury.

One lens from each rabbit with naphthalene cataract was used for the glutathione estimation, the other being used for the reduction test. In each determination a normal lens was used at the same time as a control. It was found difficult to prevent unequal agitation of the fluid due to the boiling caused by the low pressure employed, and this factor probably resulted in a certain variation in the time necessary for reduction.

The method for the determination of glutathione is far from satisfactory for such small quantities of material, as the figures for normal lenses indicate. Parallel titrations of normal and cataractous lenses, however, showed such a marked decrease of glutathione in the cataractous lenses that it is of some value.

RESULTS

The amounts of glutathione in twenty normal rabbit lenses, as expressed in per cent of lens weight, are shown in the following tabulation:

0.359	0.369	0.434	0.631	0.631
0.342	0.379	0.472	0.434	0.508
0.369	0.329	0.486	0.522	0.434
0.374	0.406	0.309	0.309	0.522
Average				0.431

The average amount found was 0.431 per cent. The higher amounts were in the later tests when 5 cc. of trichloracetic acid was used instead of 20 cc.

The table shows the amounts of glutathione found in eight lenses of rabbits with naphthalene cataract in various stages. Twelve human lenses with senile cataract removed at operation showed no glutathione.

Since these were removed by capsulotomy, they consisted chiefly of the senile nucleus with a small amount of cortex.

The sodium nitroprusside test was made with the residue left after extracting the lenses with trichloracetic acid. In the case of the human cataractous lenses, it was, as a rule, negative. With normal lenses and all the naphthalene cataracts it was positive.

Eight lenses were tested for ability to reduce methylene blue, as compared with a similar number of normal lenses. The time required varied considerably, but in all cases the cataractous lenses showed a reduction time no greater than that of normal lenses. In fact, the time required was often less with the cataractous than the normal lens,

Amounts of Glutathione in Lenses of Rabbits with Naphthalene Cataract in Various Stages

No.	Condition of Lens	Weight, Mg.	Glutathione, per Cent	No. Days Fed	No. Days Until Killed
1	Sunflower fissures, posterior pole.....	404.1 420.6	0.189	4	5
2	White soft, opaque.....	407.1 441.1	0.0225 0.0226	11	13
3	Almost completely opaque, soft.....	475.4 512.7	0.0134 0.0181	13	18
4	Completely opaque, brown anteriorly....	560.8	0.0211	13	33
5	Completely opaque, anteriorly posteriorly soft	384.0 463.0	0.0107	13	23
6	Completely opaque, hard.....	377.9	0.0262	7	114
7	Completely opaque, firm brown.....	519.9 535.1	0.0103	10	19
8	Completely opaque, brown anteriorly....	608.9 597.4	0.000856	10	25

which may possibly be explained by the fact that the cataractous lenses were swollen and soft and so allowed more intimate contact with the solution than was the case with normal lenses. The boiling due to the negative pressure could not be kept constant and allowed unequal agitation. Hence exact reports of the time required for reduction would be of little value. It can be definitely stated, however, that these opaque lenses did retain the ability to reduce methylene blue as rapidly as normal lenses.

COMMENT

The fact that glutathione was absent in human cataractous lenses was to be expected from the work of Jess and others, as was the reduction in amount of glutathione in advanced naphthalene cataract. The present figures show with fair accuracy that opacities of the lens occurred at a time when glutathione was very slightly reduced, and by the time opacity was almost complete, glutathione was reduced to less than one tenth of the amount normally present.

It would be impossible to conclude from this that the lack of glutathione causes the opacities of the lens or that the glutathione disappeared

only after the lens becomes opaque. Rather the opacities and loss of glutathione seem each to represent changes in the lens due to the ingestion of naphthalene.

The facts that a positive nitroprusside reaction was obtained in the residue of extracted lens material which contained almost no glutathione, and that the reducing power of the lens substance for methylene blue was not impaired in lenses containing such small amounts of glutathione is interesting. Goldschmidt found a positive nitroprusside reaction after all glutathione had been removed from the lens by dialysis. But such lenses did not reduce methylene blue unless a minute amount (1 mg.) of cystine was added. In the naphthalene cataracts studied in the present work, a trace of glutathione was always present, which was evidently enough to keep the reaction cysteine $\rightleftarrows$ cystine from ceasing, enough, in fact, to allow it to continue at a normal rate.

In the nuclei of human cataracts, on the other hand, the complete absence of glutathione, negative nitroprusside reaction and absence of any ability to reduce methylene blue suggests that this reaction cannot go on when all of the glutathione is absent. According to Goldschmidt, a minute amount of cystine (1 mg.) is necessary to allow the insoluble thermostable substance to undergo oxidation and continue the oxidation reduction mechanism.

In Adams' experiments on oxygen consumption in a respirometer, it was found that this process did not occur with naphthalene cataracts. Glutathione was absent in the lenses, according to her findings, as early as fourteen days after cataract developed. This differs from the present findings in which some glutathione was always found, even as late as one hundred and fourteen days after naphthalene was begun.[10] This may be explained by slight differences in technic and calculations, as the amounts found were very small and those in the normal lens were slightly higher than those found by Adams. The fact that reduction was still possible with these lenses indicates also that some glutathione was present.

The findings suggest that there is a close relationship between the lack of glutathione and death of the crystalline lens resulting in opacity. It cannot be said that lack of glutathione is the cause of opacity, however, since the early stages of opacification occurred when glutathione was still present in sufficient amounts to allow practically normal oxidation-reduction processes.

Dr. Sanford R. Gifford, Dr. G. Lowell Dunn and Dr. C. O. Miller aided in carrying out and reporting this work.

10. In the lens of rabbit 8, the amount estimated was so minute that it is possible no glutathione was present.

OPHTHALMOLOGY IN UKRAINE

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Diseases of the eye comprise approximately from 5 to 6 per cent of all diseases in Ukraine. Trachoma and occupational diseases and industrial trauma of the eye are the dominant problems that interest ophthalmologists, scientific institutions and the Ukrainian Public Health Service, because these diseases represent a large percentage of those concerned in the etiology of blindness.

Ocular diseases due to occupation and industrial trauma have increased considerably during recent years. Many factories and various new buildings have been opened in Ukraine during the last few years. This industrial development necessitates additional workers who come, untrained, from the country to the city, and their lack of acquaintance with modern machinery is the main cause of the increase in trauma and occupational diseases.

The scientific work in this branch of medicine now occupies a more important position in ophthalmologic institutions and in the ophthalmologic departments of the special new "Institutions for the Hygiene and Pathology of Labor." Trachoma is still a serious problem in Ukraine, although it has not increased in late years.

According to official medical statistics, the incidence of trachoma is 33 per ten thousand of the population; the reports of periodic inspections made by different eye institutions and hospitals show a higher percentage, especially in districts with a national minority (comprising Greeks, Germans, Bulgarians, Jews and Poles). The percentage of trachoma in some districts with a national minority is: Jews, 4.94 per cent; Greeks, 10.00 per cent; Bulgarians, 16.86 per cent, and Germans, 16.92 per cent. I believe that the causes for the spread of trachoma are the mode of living and cultural, economic and other conditions. The percentages given are similar to those computed for other parts of the world. According to the material presented at the Thirteenth International Ophthalmological Congress in Amsterdam in 1929, the great complex of social, cultural, economic and living conditions is the primary reason for the spread of trachoma all over the world.

As to the great hygienic and social problem of the status and etiology of blindness in Ukraine, the coefficient of blindness has become lower in the last few years. In 1897 blindness affected 16.7 of every 10,000

Submitted for publication, Dec. 21, 1931.

persons. According to the census of 1926, there were 31,600 blind persons, or 10.9 per ten thousand of the population. (The total population of Ukraine is 29,017,000.) This percentage is somewhat lower than that for many European and other countries; for instance, the index in England is 12 per ten thousand of the population, in Spain 11.1, in Portugal 12, in Latvia 15.4, in Esthonia 21.7, in Lithuania 22.5, in Japan 17, in Persia 24.9, in Egypt 119.7 and in Palestine 166.7.

For an analysis of the etiology of blindness there is no absolutely accurate basis, because the data and material vary; sometimes different classifications of the etiologic factors are used. Therefore, I shall give the average data for the etiology of blindness from the reports of many authors. The percentage of blindness according to cause is: trachoma, from 3.2 to 17.9 per cent; smallpox, from 3.2 to 17 per cent; trauma of the eye, from 6.5 to 24.2 per cent; blenorrhea, from 2.6 to 4.5 per cent; scrofulosis, from 3.9 to 19.1 per cent; glaucoma, from 4.3 to 9.7 per cent; diseases of the optic nerve and retinal diseases, from 7.7 to 38.4 per cent; shrinking of the eyeball and scar degeneration of the cornea, from 4 to 16.7 per cent, and intra-uterine conditions, from 3.7 to 10.4 per cent.

What is the status of ophthalmology and what aid is rendered in Ukraine at the present time? According to medical literature, little attention was paid to ophthalmology and ophthalmologic aid, especially public aid, in prewar time. Theoretical, experimental and clinical ophthalmology was inadequate in clinics, hospitals and ophthalmologic societies, and even less adequate were the study of hygienic problems in ophthalmology, the development of methods for health education and the system of preparing well trained specialists. No ophthalmologic journal existed in Ukraine; just one special ophthalmologic journal was published in Moscow for all Russia. Only from 150 to 200 oculists practiced in Ukraine, and few ophthalmic institutions existed.

Since then marked changes have taken place in ophthalmology. Ukraine now has 4 medical schools with ophthalmologic departments (Kharkow, Kiev, Odessa and Dnjepropetrovsk). A new medical school has been opened in Stalino and Lugansk, to which an ophthalmologic clinic is to be added later. Moreover, there are 4 ophthalmologic clinics which are part of institutions for the training of physicians. In addition there are a special ophthalmologic institute with 105 beds, 2 special ophthalmologic hospitals and 42 ophthalmologic departments in addition to general hospitals. Altogether, there are about 1,000 beds in all ophthalmologic institutions. All bacteriologic institutes have research laboratories for trachoma. There are now more than 500 oculists in Ukraine.

A noticeable progress in theoretical knowledge and in practical ophthalmologic work has taken place in the last few years. Many oculists from the country go to the ophthalmologic institutions for study and improvement and are paid by the Central and States Public Health Department. The teaching is now much better than formerly. Extensive scientific work is done in the bacteriologic institute in Kharkow on the etiology of trachoma (by Noguchi's method).

The Public Health Department assists the ophthalmologic institutions financially in the editing and printing of scientific works, popular literature for workers and farmers and pictures and advertisements concerning ocular diseases. Several trips to different parts of Ukraine are made by doctors every year to study the influence of various working conditions, particularly farm work, on the eye, the status of trachoma in different places with a national minority, the status and etiology of blindness and the social etiology, pathology and prophylaxis of ocular diseases.

The Ukrainian ophthalmologic journal was started in 1929, and in the same year the First Ukrainian Ophthalmological Congress was held in Kharkow. The Public Health Department organized the Central Ophthalmologic Commission, which discussed different ophthalmologic problems. In 1931 the Central Commission discussed a five year plan for practical and theoretical ophthalmology and dispensing methods, prophylaxis and education. To meet the conditions existing in public health, the following plan for theoretical and practical ophthalmology for the next few years has been worked out.

For the hospitalization of patients with ocular diseases, the following standard has been fixed: 1 bed for each 10,000 of the population in large industrial districts; 1 bed for each 15,000 in cities and 1 bed for each 2,000 in villages. Ocular treatment of outpatients will be given through ambulatory services by medico-prophylactic centers or dispensaries in cities. No payment is made for consultation, treatment, medicine, roentgenograms, etc. It is proposed to double the number of eye specialists in Ukraine in the next few years. A number of new medical buildings with modern apparatus and instruments are to be constructed. Legislative measures will be taken to protect the public from occupational diseases, trauma of the eye, etc. Protective devices for various machines, improvement in lighting conditions, protective glasses, etc., will be provided. A special ophthalmologic census of blind persons will be taken with the purpose of determining more exactly the status and the etiology of blindness. A society for trachoma is being organized and a special fund to fight the disease has been set aside by the Public Health Department.

All ophthalmologic institutions, clinics, hospitals, dispensaries and polyclinics are supplied periodically with new apparatus, instruments and laboratory equipment, produced in Ukraine or imported from foreign countries.

The Public Health Department sends doctors periodically to foreign countries for research and study of practical and theoretical ophthalmology and to ophthalmologic meetings and congresses. The questions of diseases of the eye, including glaucoma and tuberculosis, and physical methods for healing ocular diseases in which many specialists are interested are included in the program of the Second Ukrainian Ophthalmologic Congress, which will take place in 1933.

Clinical Notes

TWO DEVICES FOR THE OPHTHALMOSCOPE

D. Hamilton Row, M.D., Indianapolis

A description of two adaptations to the May Model of the Bausch and Lomb ophthalmoscope follows.

First to be described is a system of apertures drilled through from the face of the head of the ophthalmoscope into the light chamber. As shown in figure 1, the opening in the face of the instrument is just

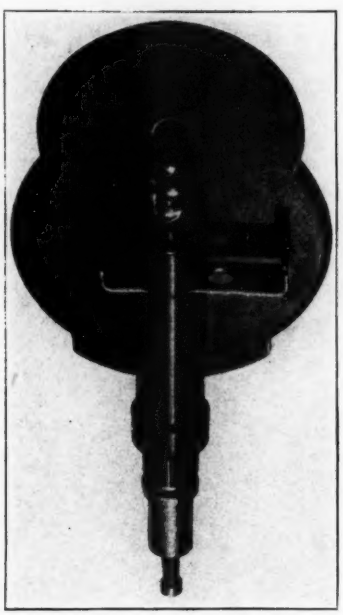

Fig. 1.—The diaphragm slide moved to the extreme right. A small field is available for examination, since the small opening of the slide is directly over the lamp of the ophthalmoscope. To the right of the light chamber of the ophthalmoscope may be seen the large opening of the slide diaphragm.

above the center pin, which acts as a pivot for the lens carrier. One hole is drilled through into the light chamber with the aperture or zero lens in position. Thus, when the zero lens comes into position for use, a small round light presents itself through the drilled opening in the face of the ophthalmoscope.

Submitted for publication, Jan. 25, 1932.

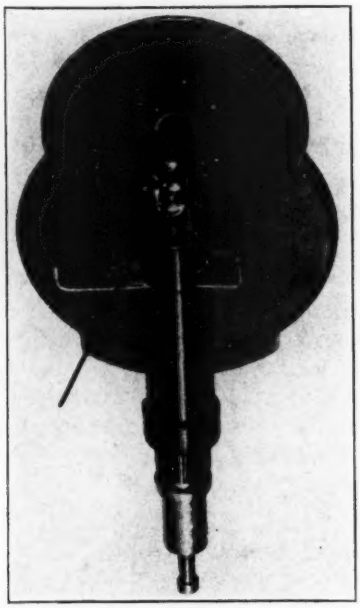

Fig. 2.—The diaphragm slide moved to its other extreme of motion to the left. A large full field is now available for examination, since the large opening of the slide diaphragm is directly over the lamp of the ophthalmoscope. The arrow points to the small diaphragm that was in position for use in figure 1.

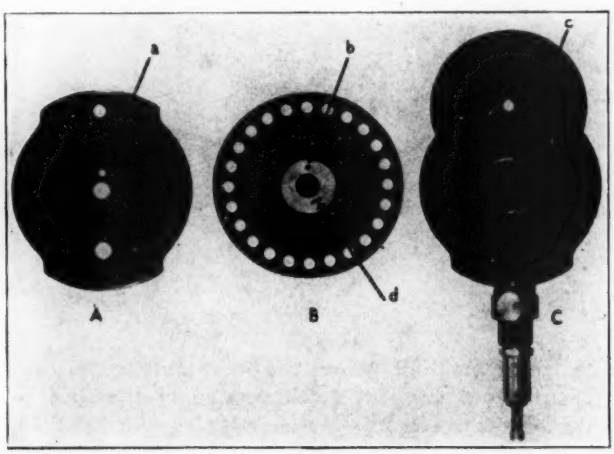

Figure. 3.—The system of apertures for locating the zero lens and the minus 20 diopter lens with only the ophthalmoscope itself lighted and the room dark. In assembling the ophthalmoscope, *B* fits over *C* and *A* over *B*. When this is done, openings *a*, *b* and *c* are in alinement, and since opening *c* is drilled through into the light chamber, a round light shows at *a* and one knows that the zero lens is in position for use. If after the assembly of *B* on *C* and *A* on *B*, *B* is revolved until point *d* assumes the present position of point *b*, there will be a band of light showing through *a*, and one knows that the —20 diopter lens is in position for use.

There is also a square-cut hole in the lens carrier unit which alines itself with the drilled opening when the —20 diopter lens is in position for use. There are, then, two points approximately opposite each other on the circuit of the lens carriage to which one can turn knowingly without the inconvenience of referring to a source of light other than the light furnished by the lamp of the ophthalmoscope. In making this device, it was found necessary to fasten the washers, which are on either side of the lens carrier, firmly to the carrier, but this seems in no way to interfere with the racking of the carrier.

The second device has to do with the size of the field under observation. A small diaphragm to slip over the condensing lens directly over the ophthalmoscope lamp has been furnished by the manufacturers for some time, and when in position it is an aid in examining small areas of the fundus and is particularly helpful in observing the fundus through a small pupil, much scleral, corneal, iridic and other reflection being eliminated. However, such a small field is not desired except in specific cases, so that heretofore it has been necessary to remove the head of the ophthalmoscope to make the change from the small to the large diaphragm, or vice versa.

By having a slot cut through the light chamber from side to side in a horizontal plane, a slide is now fitted into the slot, which slide can be moved from side to side so that when it is at one extreme of its movement a small diaphragm opening is in position for use, corresponding to the small diaphragm in common use. When the slide is at its other extreme of movement, a large full-sized field is available. The change from one to the other can be made instantly without removing the head of the ophthalmoscope.

Both of these devices have proved helpful to me in the routine use of the ophthalmoscope and add considerably to the flexibility of the instrument.

WHY DO LACRIMAL FISTULAS, PHLEGMONS AND MUCOCELES ALWAYS APPEAR BELOW THE HORIZONTAL AXIS OF THE EYE?

J. M. WEST, M.D., ALLENTOWN, PA.

It is a well known fact that when progressive inflammatory conditions of the tear sac advance sufficiently to change the normal contour of the region of the internal canthus, the swelling (phlegmon or mucocele) or fistula is always to be found below the horizontal axis of the eye (fig. 1). Even in phlegmonous cases in which the inflammation may extend above the eye the greater part of the swelling is always distinctly beneath the horizontal axis. In the literature, however, I find no exact explanation of this clinical fact, although it is constantly made use of in differentiating fistulas of the ethmoid and frontal sinuses from

Submitted for publication, Jan. 28, 1932.

From a paper read before the Philadelphia Laryngological Society, Jan. 5, 1932.

Fig. 1.—Mucocele of the tear sac in the typical position below the horizontal axis of the eye.

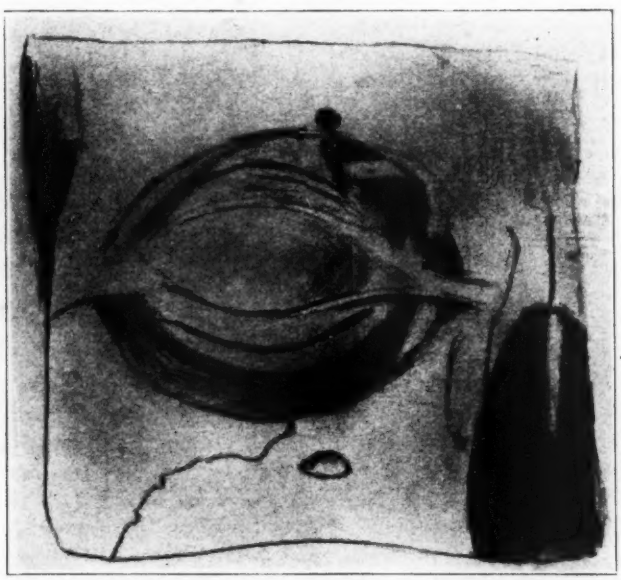

Fig. 2.—Figure showing the anatomic relations at the internal angle of the eye. It will be seen that the much greater part of the lacrimal sac is below the ligamentum palpebrale mediale. (Redrawn from Spalteholz, W.: Handatlas der Anatomie, Leipzig, S. Hirzel, 1903, vol. 3, p. 795, fig. 870.

those of the lacrimal sac. Lacrimal fistulas perforate below the horizontal orbital axis, while fistulas of the frontal and ethmoid sinuses appear above this axis.

An abscess always advances along the path of least resistance, and just as a psoas abscess follows the lumbar muscles, so there is a definite anatomic reason for inflammations of the lacrimal sac pointing below the horizontal axis of the eye.

In suppuration of the tear sac, it is the anatomic relation of the internal ligament to the sac that determines the position of the swelling or perforation when inflammatory conditions of the sac approach the skin. The mechanics of the process is to be explained in the following manner.

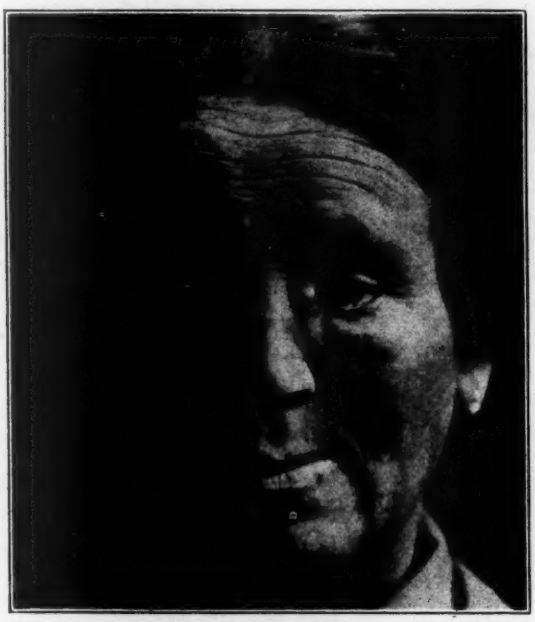

Fig. 3.—Mucocele of the remaining fornix after external extirpation of the sac. It will be noticed that the swelling is situated higher than in cases in which operation has not been performed.

The lacrimal sac embedded in the lacrimal fossa, projects about 2 mm. above the ligamentum palpebrale mediale, which stretches across its upper part from the crista lacrimalis posterior to the frontal process of the superior maxilla. Below, however, the sac extends some 8 mm. below the ligament [1] (fig. 2). Thus it is evident that while the upper part of the sac is protected by the ligament, which resists pressure from within, its lower and much greater part has no support, being covered only by the skin and subcutaneous tissue. Hence increasing pressure from within the sac will always cause it to bulge and finally perforate

1. Onodi, A.: Die Beziehungen der Tränenorgane zur Nasenhöhle und zu ihren Nebenhölen, Berlin, 1913.

in its lower segment, that is, below the ligament, which represents the horizontal axis of the eye.

Some years ago, in a paper [2] describing the results of the intranasal operation on the lacrimal sac, I called attention to the clinical significance of the internal ligament with reference to the position of lacrimal fistulas and mucoceles, but did not, however, at that time describe the mechanics of the process.

There is, however, one exception to the general statement that suppurative conditions of the tear sac always bulge and perforate below the internal ligament. Sometimes after an unsuccessful attempt to extirpate the sac externally, the fornix may not have been removed. Under these conditions a subsequent dilatation of the remaining fornix is likely to appear slightly above the horizontal axis (fig. 3). Cure can be obtained in these cases by the intranasal operation on the tear sac, in just the same way as healing is obtained in those on which external operation has not been done previously.

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2. West, J. M.: Arch. f. Laryng. u. Rhin. **30**:215, 1916.

Ophthalmologic Review

EDITED BY DR. FRANCIS HEED ADLER

CHEMICAL BURNS OF THE EYE

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A chemical burn of the eye is, without a doubt, a serious condition. Because of the rapid action of the chemical, its destruction of tissues and its penetrating power, and because of the fact that a chemical burn does not respond to treatment, it is a challenge to every ophthalmologist. One needs but to see one case of this type to realize the truth of this statement. A chemical burn of the eye is much worse than a burn by fire or hot metal.

Reported cases are not frequent. Wagenmann,[1] in the Graefe-Saemisch Handbuch, reported twenty-three cases up to 1911. Teräskeli [2] in 1927 could find only twenty-four cases in the literature. What the cases lack in frequency, however, is overshadowed by their disastrous results.

A chemical burn, according to Thies,[3] is a change in the bodily tissues produced by the entrance of the chemical substance. The action of the chemical on the different parts of the eye is different, as the coats of the eye have their own peculiar structures. The susceptibility increases as one goes from the skin to the conjunctiva and the cornea.

The action of a chemical on the eye depends on the following points: the strength of the chemical, the quantity reaching the eye, the peculiar chemical construction of the substance, the duration of its action and the pressure under which it strikes the eye. In this review, burns caused by alkalis and acids will be considered.

ALKALIS

Ammonia.—Burns of the eye due to ammonia are the most serious of all chemical burns, and for this reason will be considered first. Liquid ammonia, on striking the eye, causes an intense edema of the lids, marked conjunctival congestion and chemosis, cloudiness of the cornea, often followed by hypopyon, a contracted pupil with a posterior synechia, cataract and panophthalmitis. The cloudiness of the cornea is never as dense as that seen in lime or lye burns. If the cornea is anesthetic after the injury, a poor prognosis is assured, according to Denig [4] and Wagenmann. A peculiar feature about ammonia burns,

which has been observed by all writers on this subject, is that, while the immediate prognosis may appear favorable, it is not until about the eighth or tenth day that a distressing change takes place. The cornea begins literally to melt away, panophthalmia develops and the eye is lost. This feature was emphasized by Thies,[5] Pilcher,[6] Hoffmann,[7] Trousseau[8] and Denig. One should bear this fact in mind in making a prognosis.

The pathologic action of ammonia and alkalis on the eye is entirely different from that of acids and lime. According to Thies,[9] Wagenmann,[1] Lewin and Guillery[10] and Pilcher,[6] ammonia acts as a base on living and dead albumin, forming a soluble alkali albuminate which possesses great toxic power. This albuminate appears as a soft eschar, possessing a high degree of affinity for water. On account of this water-extracting power, especially in the case of ammonia, the alkali penetrates deeper into the soft tissues, causing more damage and greater necrosis. Since the cornea contains about 80 per cent water, according to Leber, as soon as the ammonia strikes it, a cloudiness develops as a result of physical changes in the tissues, such as water extraction and vacuole formation. The molecular structure of the cornea is disintegrated, the alkali penetrates deeper and the cornea slowly melts away (Einschmelzung). During the first week following the injury, the eye seems to be improving, and one may falsely anticipate a favorable result. It is only after the first week that the climax is reached. A decided change for the worse is noted and the eye is rapidly destroyed. This is the usual course of events in an alkali burn of the eye. In the case of a burn due to ammonia gas, the eye is invariably lost, according to Thies[3] (1928) and Lewin and Guillery.[10]

The action of lye resembles that of ammonia, but it is less severe (Thies[11] and Guillery[12]).

Only recently Thies (1931) reported the case of an ammonia burn of the eye in which an oral mucous membrane graft was used on the eyeball. The patient did well until the seventh week, when a relapse occurred with necrosis of the graft. Thies explained this relapse by stating that some of the ammonia was still present in the lower fornix of the eye and had not been completely removed. This explanation is doubtful. The case, however, emphasizes the seriousness of an ammonia burn of the eye. Thies also warned against a favorable prognosis, even though there is a regression of the inflammatory changes in the tissues.

Even spirits of ammonia, which is a common household remedy containing about 10 per cent ammonia, if allowed to come in contact with the eye can destroy it, as in the case reported by Abramovicz.[13]

Acid Burns.—The action of acid differs greatly from that of an alkali in burns of the eye. Acids precipitate the albumin of the conjunctiva and cornea and form dense scars. This formation of dense

scars or eschars stops any further action of the acid, and there is no penetration of the acids into the deeper tissues. There is no prolonged action of the acids on the tissues as seen in the case of ammonia. The entire damage has been completed in a few seconds. Acids do not possess the penetrating power of ammonia and alkalis (Guillery [14]).

Lime Burns.—In lime burns of the eye, according to the researches of Guillery,[10] Zur Nedden [15] and Fuchs,[16] the corneal opacity is due to a chemical combination of the lime with the cornea, forming calcium albuminate and calcium carbonate. As the affinity of lime for albumin is slight in comparison with its affinity for carbonic acid, calcium carbonate probably forms the major part of the opacity. This is the opinion of the Barkans [17] (1924). The gradual increase in the opacity is due to the further conversion of the albuminate into the carbonate by the carbonic acid of the tissues and the air. The opacity is really due to an incrustation of calcium carbonate.

EXPERIMENTAL WORK

Guillery,[14] in his experiments on animals, showed that ammonia never produces the dense corneal cloudiness seen in lime or lye burns.

Hoffmann [7] performed experiments on fresh corneas of oxen to test their diffusion power with ammonia. He also used the corneas of live rabbits. Within seven minutes, the ammonia had already passed into the anterior chamber. The greater the concentration of the ammonia, the more rapid is its action. Hoffmann believed that the formation of cataract in cases of ammonia burns of the eye is not a result of the direct action of the ammonia but rather of an indirect action.

Siegrist [18] performed experiments on rabbits' eyes using ammonia in one eye and concentrated nitric acid in the other eye, and studied the results. The ammonia destroyed the entire cornea in the shortest time and exercised a direct inflammatory action on the iris and ciliary body. The acids caused a coagulation of the superficial layers of the cornea, the deeper layers remaining unchanged. No primary iritis developed. The capsule of the lens can be injured by the diffusion of the acids through the cornea. Strong acids will also affect the iris. Siegrist believed that it is possible for the chemical substances in small doses, by diffusing through the cornea, to affect the iris and produce iritis.

Kuwabara [19] attempted to prove experimentally the relation of ammonia to the formation of cataract in human beings. Working on animals, he found that very weak solutions of ammonium salts and ammonia, even 1 : 10,000 dilutions, produce opacities of the lens and cataract.

Yoshimoto [20] performed experiments on rabbits' eyes, using acids and alkalis. He used sodium hydroxide, ammonium hydroxide, hydrochloric acid, sulphuric acid, nitric acid and acetic acid in different concentration. He began with tenth-normal and gradually increased their concentration up to third-normal. The order of the chemicals in degree of severity was as follows: soda lye, hydrochloric acid, sulphuric acid, nitric acid, acetic and ammonium hydroxide. Burns with the first three solutions of a normal concentration caused phthisis of the eye. The second three solutions in a third-normal concentration caused phthisis bulbi. After soda lye burns, the formation of phthisis bulbi could be stopped as late as five minutes after the injury by a thorough flushing of the culdesac with acidulated water or a 1 per cent solution of tannic acid if the concentration of the lye was not more than half-normal. After acid burns, phthisis bulbi and leukomas could be stopped if one used copious irrigations of the sac with a mild alkaline water of a 1 per cent solution of tannic acid if the concentration of the acid was not more than normal. Anatomic examination of the tissues showed great differences, both quantitatively and qualitatively, between an acid and an alkali burn. Yoshimoto stated that a lye burn is intensive, and repair processes appear late. In an acid burn repair comes on much earlier.

TREATMENT

The treatment for chemical burns of the eye is not specific. Some regard a case of ammonia burn as hopeless (Seefelder [21] and Gerard [22]). Many ophthalmologists attempt to neutralize the substance in the eye by using another chemical. Such a procedure is against the experimental work of several writers. Cosgrove and Hubbard,[23] from their experiments on rats and rabbits in the treatment for chemical burns produced by sulphuric acid, nitric acid, phenol, sodium hydroxide and ammonium hydroxide of different concentrations, stated that regardless of the concentration of the chemical or the time interval, the best results are obtained by first using copious irrigations of water. Any attempt at neutralization actually causes more damage. Mechanical removal of the chemical, especially in the case of phenol, is the most important factor. The time that elapses before treatment is given is an important point. After a certain lapse of time, with any concentration, irreparable damage is done.

Davidson,[24] from his experiments on rats with acid and alkali burns of the skin, stated that washing the injured area with water to remove the excess chemical is much better than attempting to neutralize the area. Only after prolonged irrigations with water should neutralization be attempted. Pilcher [6] expressed the same opinion.

In the case of ammonia burns, Siegrist,[18] Thies [25] and Pilcher [6] recommended puncture of the anterior chamber to remove any ammonia present in the aqueous.

Denig's [26] earlier experience with ammonia burns was unsatisfactory. In 1920 and 1927, he wrote that immediate surgical intervention in the form of oral mucous membrane grafts offered the best method of treatment. He believed that this procedure will save the cornea when there is any danger of necrosis due to destruction of the surrounding conjunctiva. Rötth [27] believed that Denig used the plastic operation for burns too early. His own indications for plastic operations are: 1. If the greater part of the conjunctiva has sloughed away. (If there is any suspicion of necrosis in the superficial layers of the sclera, the results are poor.) 2. If the cornea shows loss of epithelium and is insensitive. Thies [28] wrote a paper on the question of when to operate. He believed in early operation but not as early as Denig recommended. In six of his cases in which operation was done before the fifth day, the results were good.

Hurst [29] prevents the formation of symblepharon by stitching back the lids away from the globe onto the forehead and cheek. Colrat [30] attempts to prevent the formation of symblepharon by frequent movements of the lids. Wagenmann [1] had no specific treatment to offer.

Thies [3] (1928), who has had an extensive experience in these cases, advised the following procedure: The conjunctival sac should be carefully cleansed of all the chemical by copious irrigations of water. The débris and dead tissue should be removed. A sterile ointment is instilled and a bandage applied. He makes no attempt to prevent the formation of symblepharon, as he believes that it is impossible. For exuberant granulation, he recommends the galvanocautery. When healing has come to a standstill, operation is indicated.

Stieren [31] believes that atropine is contraindicated in ammonia burns on account of the danger of secondary glaucoma. Bentzen,[31] by experiments on ammonia burns in animals, showed that there was an increase in the intra-ocular tension. Lieb, in 1912, reported a case of glaucoma following an ammonia burn of the eye. None of the textbooks written on this subject make any reference to the use of atropine in chemical burns of the eye.

Cross [33] recommended the use of glycerite of tannic acid for ammonia and alkali burns. He said that in his experience this drug stopped the prolonged action of the chemical.

In the treatment for corneal opacities following lime burns Barkan [17] recommended the use of neutral solutions of ammonium tartrate beginning with 4 per cent twice daily. Zur Nedden [15] had already advocated this remedy. The eye should first be cocainized before the

ammonium tartrate is used. The solution must also be neutral. Barkan used this treatment in four cases with fair results. Wolff [34] also recommended this treatment for clearing corneal opacities.

CONCLUSIONS

1. Ammonia burns of the eye are the most serious of all the chemical burns.

2. The prognosis should be guarded, on account of the danger of a relapse.

3. There is no specific treatment for chemical burns of the eye.

4. Copious irrigations with water are better than chemical neutralization.

5. Instillations of glycerite of tannic acid should be tried.

6. Denig's method of oral mucosa graft should be used more frequently.

7. Neutral solutions of ammonium tartrate are of value in clearing corneal opacities following lime burns.

BIBLIOGRAPHY

1. Wagenmann, in Graefe-Saemisch: Handbuch der gesamten Augenheilkunde, Leipzig, Wilhelm Engelmann, 1911, vol. 9, pt. 2, p. 1531.
2. Teräskeli, H.: Acta ophth. **4**:274, 1927.
3. Thies, O.: Arch. f. Augenh. **99**:188, 1928; Klin. Monatsbl. f. Augenh. **82**:353, 1929.
4. Denig, R.: Ztschr. f. Augenh. **11**:308, 1904; Am. J. Ophth. **3**:256, 1920.
5. Thies, O.: Klin. Monatsbl. f. Augenh. **72**:378, 1924; **79**:534, 1927.
6. Pilcher, A.: Ztschr. f. Augenh. **23**:297, 1910.
7. Hoffmann, V.: Arch. f. Augenh. **91**:300, 1922.
8. Trousseau: Ann. d'ocul. **113**:55, 1895.
9. Thies, O.: Arch. f. Ophth. **123**:165, 1929.
10. Lewin and Guillery: Die Wirkungen von Arzneimitteln und Giften auf das Auge, Berlin, A. Hirschwald, 1913, vol. 2, p. 792.
11. Thies, O.: Arch. f. Ophth. **115**:346, 1925.
12. Guillery: Arch. f. Augenh. **53**:258, 1909.
13. Abramovicz, I.: Brit. J. Ophth. **9**:244, 1925.
14. Guillery: Arch. f. Augenh. **56**:221, 1907
15. zur Nedden: Arch. f. Ophth. **63**:319, 1906.
16. Fuchs: Ber. ü. d. Versamml. d. deutsch. ophth. Gesellsch., Heidelberg, 1905, p. 225.
17. Barkan, H., and Barkan, O.: Treatment of Lime Burn of the Eye, J. A. M. A. **83**:1567 (Nov. 15) 1924.
18. Siegrist, A.: Ztschr. f. Augenh. **43**:176, 1920.
19. Kuwabara, T.: Arch. f. Augenh. **69**:256, 1911.
20. Yoshimoto: Arch. f. Augenh. **99**:188, 1928.
21. Seefelder: Klin. Monatsbl. f. Augenh. **78**:82, 1927.
22. Gerard: Clin. opht. **15**:251, 1926.

23. Cosgrove and Hubbard: Ann. Surg. **87**:89, 1928.
24. Davidson, E. C.: Ann. Surg. **85**:481, 1927.
25. Thies, O.: Klin. Monatsbl. f. Augenh. **79**:534, 1927.
26. Denig, R.: Arch. f. Ophth. **118**:729, 1927; Am. J. Ophth. **3**:256, 1920.
27. Rötth, A.: Klin. Monatsbl. f. Augenh. **82**:669, 1929.
28. Thies, O.: Arch. f. Ophth. **120**:689, 1928.
29. Hurst, V. R.: Texas State J. Med. **20**:41, 1924.
30. Colrat: Arch. d'opht. **46**:416, 1929.
31. Stieren: Ophth. Rec. **13**:494, 1904.
32. Bentzen: Arch. Ophth. **41**:65, 1895.
33. Cross: Personal communication to the author.
34. Wolff: Brit. J. Ophth. **10**:196, 1926.

Correspondence

DIPLOPIA

To the Editor.—In the article on "Diplopia and Other Disorders of Binocular Projection" by the late Dr. Duane in the February issue of the ARCHIVES OF OPHTHALMOLOGY (**7**: 187, 1932), should not the phrase on page 191, line 2 from the bottom, read, "10 degrees *below* the point" instead of as given "10 degrees above the point"? There is also, it seems to me, some difficulty in following the discussion on the torsional diplopia because the definitions are not given fully.

From a careful analysis, it seems that Dr. Duane calls "intorsional diplopia, DI," an intorsion of the *lower* parts of the vertical images, that is, the projected images converge below like the two limbs of the letter V. Similarly, he calls "extorsional diplopia, DE," an extorsion of the *lower* parts of the vertical images, that is, the projected vertical images diverge below like the two limbs of the letter A. However, in referring to the torsion or tilting of the eye itself, he refers to the *upper* part of the vertical meridian, so that when he speaks of both vertical meridians of the eyes *turning in,* he means that the *upper* parts of these meridians turn in and converge above like the two limbs of the letter A. Similarly, in referring to the vertical meridians of the eyes turning out, he apparently refers to the *upper* parts of these meridians, which tilt outward and diverge above like the two limbs of the letter V.

The terms "intorsion" and "extorsion" as generally used refer to the *upper* parts of either the vertical meridian or the vertical image. Intorsion refers to the *upper* part of either one turning nasalward, or toward the median plane; extorsion refers to the *upper* part of either one turning outward, temporalward, or away from the median plane. Dr. Duane uses these terms for the images, but apparently in just the opposite sense. For the torsion of the meridians of the eye, he uses the term, "contorsion" for what it seems is usually referred to as intorsion, or perhaps here more properly, incycloduction, and the term, "distorsion," for what is usually known as extorsion, or perhaps here more properly, excycloduction.

The foregoing is what I have worked out after some analysis of the article, and I believe it to be correct. However, perhaps some editorial clarification of this splendid article aside from the correction of some errors would make it more understandable.

JOSEPH I. PASCAL, M.D.

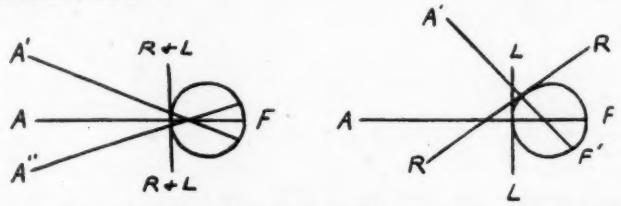
To the Editor.—In answer to Dr. Pascal's letter, I think a few words more regarding torsional diplopia would have cleared up any question; however, it was probably so clear to Dr. Duane that he did not see a chance for misunderstanding. To begin with, a tilting of the vertical meridian of the cornea to Dr. Duane always meant a tilting of the upper end of the meridian.

Considering the table of abbreviations on page 188 of this article, when the right image is below, it is a DR (right eye higher). When the left image is below, it is a DL (left eye higher). Therefore, reasoning along the same line with B, when the upper end of the image is tilted out, the eye is tilted in, and so is a DI. When the upper end of the image is tilted in, the eye is tilted out, and so is a DE.

I do admit that it is confusing, and we should aim to clear up chances of misinterpretation, that is, to say definitely whether we are speaking of the upper end of the vertical meridian of the cornea or of the diplopia produced by the tilting. There is common confusion in speaking of vertical and torsional diplopia.

The only reason we have less confusion in the lateral diplopia is because we are more used to them, but in speaking of a homonymous diplopia, the visual axes are crossed, and in crossed diplopia, the visual axes are not crossed. Explained by the theory of the binoculus, they all become more plain.

The question relating to the vertical deviation of 10°, is best shown by the binoculus, as:



In the sketch, *1* is the binoculus with both eyes coinciding. In *2*, however, *L-L* is a line tangent to the cornea and at a right angle to the visual axis of the left eye. *F* indicates the fovea and *A*, the point of fixation. The line *R-R* is a line tangent to the cornea of the right eye and at a right angle to the visual axis. The object *A* is 10° above *F'*, or the fovea of the right eye, and so is projected 10° below the image *A*, seen by the left eye.

Regarding the naming of torsions and ductions, I do feel that some nomenclature should be decided on, and then adhered to.

JAMES W. WHITE, M.D.

News and Notes

EDITED BY DR. JOHN HERBERT WAITE

SOCIETY NEWS

Oxford Ophthalmological Congress: Preliminary Notice.—The Oxford Ophthalmological Congress will assemble at Keble College, Oxford, on the evening of Wednesday, July 6, 1932, and the meetings will be held on the following Thursday, Friday and Saturday, July 7, 8 and 9. On the morning of Thursday, July 7, there will be a discussion on "The Treatment of Nonparalytic Strabismus Excluding Operative Details," to be introduced by Dr. Luther Peter, of Philadelphia. The following have already signified their intention to contribute to the discussion: Mr. Bernard Chavasse (Liverpool), Mr. Arthur Ormond (London), Mr. Edward Alabaster (Birmingham), Mr. F. Williamson-Noble (London), Dr. Stewart Barrie (Glasgow) and Mr. George Young (Colchester). The Doyne Memorial Lecture, entitled "The Ocular Signs in the Phakomatoses of Bourneville, von Hippel-Lindau and Recklinghausen," will be delivered on the morning of Friday, July 8, by Professor van der Hoeve (Leiden). Dr. Canti (London) will give a cinematograph demonstration of living cells and the influence of radium on malignant cells. In addition to the foregoing, the following papers have already been promised: Professor Dr. Patcheff (Sofia), "The Laws of True Trachoma," with demonstrations; Mr. J. Burden-Cooper (Bath), "Some Observations in the Practice of Refraction." There will probably be a demonstration of "The Thomas Photoelectrographe." This is an apparatus enabling the blind to read newspapers, books, typewriting or handwriting, reproduced in relief through a system of photo-electric cells which cause a number of thin metallic rods to shape the letter as in Braille type. There will also be demonstrations in the Scientific and Commercial Museums, to which members are invited to bring forward novelties of any ophthalmologic interest. It will be much appreciated if those members who desire to contribute papers, discussions or demonstrations will be good enough to notify the secretary at their earliest convenience. Ophthalmologists desirous of admission to membership in the Congress are requested to communicate with the secretary, who will send application forms for membership and full details on request. There is no entrance fee or annual subscription, but a contribution of 2 pounds, 2 shillings is payable on each occasion that a member attends a congress. The annual dinner of the congress will take place in the hall of Keble College on Thursday, July 7, at 7:45 p. m., to which guests (ladies or gentlemen) introduced by members will be welcome; charge, 15 shillings. The Master and Mrs. Cyril Walker will receive members and their guests in the hall of the college, the first portion of which will be

arranged as a reception room. On Thursday afternoon there will be tea at New College, where members and their friends will be received by Professor and Mrs. Townsend. A general meeting of the members will be held during the period of the congress. As before, accommodation will be available in Keble College, the tariff being 10 shillings, 6 pence a day, made up as follows: rooms, 3 shillings; breakfast, 2 shillings; lunch, 2 shillings, 6 pence, and dinner, 3 shillings. Members not residing in the college may obtain meals in the hall at the foregoing rates, for which payment should be made at the time. It is hoped to be able to secure college or other accommodation for lady members, wives of members or other lady friends. Members will meet informally at dinner on Wednesday, July 6, at 7:45 p. m., in the hall of Keble College. The full program of the congress will be issued in June.

C. G. Russ Wood, *Hon. Secretary and Treasurer,* Hill House, Abberbury Road, Iffley, Oxford, England.

Ophthalmological Society of the United Kingdom.—The Annual Congress of the Ophthalmological Society of the United Kingdom will be held at the Department of Physiology, the University of Edinburgh, on May 12, 13 and 14, 1932. The program is as follows: May 12: 9:30 to 10:30 a. m., opening address, Dr. A. H. H. Sinclair, president. 10:30 a. m. to 12:30 p. m., Dr. Josef Urbanek, "Ocular Tuberculosis"; Mr. David Lees, "Observations on the Use of Tryparsamide in the Treatment of Optic Atrophy Due to Syphilis"; Mr. Maurice Whiting, "Syphilitic Tarsitis"; Dr. H. M. Traquair, "External Dacryorhinocystostomy." 2:15 to 4:15 p. m., Mr. Stephen Mayou, "Congenital Absence of the Corneas in a Foal," and "Report on a Case of Blood-Staining of the Eye"; Mr. C. Edmund, "Nyctalopia and Hemeralopia"; Dr. Clark Souter, "Papillomatosis Conjunctivae: Spontaneous Cure of a Case"; Mr. Charles Goulden and Mr. H. B. Stallard, "Two Cases of Epibulbar Squamous Cell Carcinoma." 5 p. m., Prof. Dr. J. van der Hoeve, Bowman Lecture, "Ocular Movements." 7:15 to 7:45 p. m., annual dinner of the society at the Royal College of Surgeons. May 13: 9:30 a. m. to 12 noon, discussion on "Senile Cataract: The Preoperative and Postoperative Management and Complications." Opener, Dr. A. J. Ballantyne, followed by Sir Arnold Lawson. The following have also intimated their desire to take part in the discussion: Mr. H. T. Holland, Dr. H. M. Traquair, Mr. A. F. MacCallan, Mr. R. Foster Moore, Lt.-Col. A. E. J. Lister, Lt.-Col. R. E. Wright and Mr. W. B. Inglis Pollock. 12 noon, Mr. G. F. Alexander, "The Lip Graft Operation in Trichiasis." 2:15 to 4:15 p. m., clinical cases, Eye Department, Royal Infirmary. 5:15 p. m., demonstration in the museum, by Dr. Black, of Miklos' Perimeter and Apparatus for Localizing Tears in Detachment of the Retina. 8:30 p. m., Mr. Ernest Thomson, "The Organization and the Administration of School Ophthalmic Clinics in the County of Lanark"; Mr. John Marshall and Dr. C. W. Graham, "Blindness and Its Certification." May 14: 9:30 a. m. to 12 noon, Mr. C. D. Shapland, "Retinal Detachment and Gonin's Operation: A Statistical Survey; Some Observations on Bilateral Symmetrical Detachment of the

Retina"; Mr. Norman M. Dott, "Two Cases of Meningioma of the Tuberculum Sellae, Illustrating the Value of Ophthalmologic Examination"; Mr. J. Tudor Thomas, "Transplantation of Scleral Tissue on to Rabbit's Cornea"; Mr. E. Wolff, "A Case of Recurrent Proptosis": Mr. W. B. Inglis Pollock, "Report on Two Cases of Cerebro-Retinal Familial Degeneration Reported as Two Cases of Leber's Disease (*Tr. Ophth. Soc. U. Kingdom* 37: 247, 1917)"; Mr. Norman M. Dott, "Intracranial Resection of the Optic Nerve in Glioma Retinae."

Council of British Ophthalmologists.—The annual report of the Council of British Ophthalmologists will be presented at a meeting to be held after the business meeting of the Annual Congress of the Ophthalmological Society of the United Kingdom, Edinburgh, Saturday, May 14, at 12: 30 p. m. All British ophthalmic surgeons are invited.

Courses for Medical Social Workers.—The National Society for the Prevention of Blindness plans to sponsor, next fall, two courses for medical social workers in eye hospitals. The society will cooperate with the Massachusetts Eye and Ear Infirmary, Boston, and with Washington University, St. Louis, in these courses, which will probably extend through a period of sixteen weeks. Communications should be addressed to Miss Eleanor Brown, National Society for the Prevention of Blindness, 450 Seventh Avenue, New York, relative to the qualifications for admission to these courses and the aids available.

Association for Research in Ophthalmology.—On May 10 at New Orleans, the Association for Research in Ophthalmology will provide the following program: "Eystrain and Illumination," Dr. Walter B. Lancaster, Boston; "The Principle of Induction in Color Vision," Dr. Frank Allen, Winnipeg, Canada; "The New Science of Seeing," Dr. M. Luckiesch, Cleveland; "Light Sense," Dr. Percy W. Cobb, St. Louis; "Ultraviolet Light in Ophthalmology," Dr. Rommel Hildreth, St. Louis, and "The Transmissive Properties of Tinted Lenses," Dr. W. W. Coblenz, Washington, D. C.

Colorado Congress of Ophthalmology and Oto-Laryngology and Summer Graduate Course.—The tenth annual summer graduate course and the Colorado Congress of Ophthalmology and Oto-Laryngology will be held from July 18 to 30, inclusive. All ethical practitioners are eligible for the course. The class will be divided into small sections for the demonstrations and clinics. The daily luncheons, followed by round table discussions, will be devoted alternately to ophthalmology and otolaryngology. Applications for the graduate course, accompanied by a check for $50, the total fee, should be sent to Dr. Harry L. Whitaker, treasurer, 1612 Tremont Street, Denver. The course is arranged for those interested in both ophthalmology and otolaryngology. Those who wish demonstrations only on ophthalmology or only on otolaryngology should indicate such preference when making application. Arrangements will be made to fill the demonstration hours with the special branches desired.

The lectures will be given in the auditorium of the Capitol Life Building, Sixteenth and Sherman Streets, daily from 10 to 12 a. m., and

from 2 to 4 p. m. In ophthalmology, the lectures will be as follows: "Injuries to the Eye and Related Parts, Their Early Management and Late Results," Hans Barkan, Stanford University Medical School; "Fields of Vision, Technic of Examination, Records, Visual Pathways and Their Lesions, and Field Changes in Glaucoma and Other Diseases," Cyrus W. Rutherford, University of Iowa Medical School; "Basic Optics, Defects of Refraction, and Their Measurement and Correction," Edward Jackson, Denver; "Use of Medical Literature in Ophthalmology," William H. Crisp, Denver; "Tumors of the Orbit," William C. Finnoff, Denver.

Demonstrations will be given between the hours of 8 and 10 a. m. daily. The class will be divided into sections of suitable size, and the demonstrations will be held in the various hospitals throughout the city and elsewhere as designated. The demonstrations in Ophthalmology will cover the following subjects: Principles and Use of the Cross-cylinder, Anatomy of the Retina with Slides and Photographs, Intracapsular Extraction of Cataract, Measurement of Refraction, the Slit-Lamp Corneal Microscope, and Clinical Ophthalmoscopy. These will be given by William H. Crisp, James M. Shields, William M. Bane, Donald H. O'Rourke, M. E. Marcove and R. W. Danielson.

The congress will be held Friday and Saturday, July 22 and 23, when special papers will be presented and discussed. After the program on July 22 there will be a motor trip into the mountains followed by a complimentary banquet.

Cleveland Ophthalmological Club.—At the April meeting of the club, Dr. M. Luckiesh of the General Electric Company spoke on the subject, "Seeing, a Partnership of Lighting and Vision," and paid a tribute to the pioneer work of Dr. Percy Cobb.

Colorado Ophthalmological Society.—The thirty-third anniversary of the organization of the Colorado Ophthalmological Society was signalized by a dinner meeting, given in honor of Dr. Melville Black and attended by thirty-six members and guests. Of the five original members, three were present: Drs. W. C. Bane, Edward Jackson and Melville Black. The other original members not in attendance were Drs. Edmund W. Stevens and David H. Coover. The scientific address, "The Nature of Light," was given by Prof. O. C. Lester, head of the Department of Physics, the University of Colorado.

German Ophthalmological Society.—The society will meet in Leipzig on May 19 to 21, 1932. An extensive program, comprising more than seventy papers by well known ophthalmologists and thirty-five demonstrations, has been arranged covering practically every phase of ophthalmology.

GENERAL NEWS

The Certification of Blindness.—This is the title of a report by the Prevention of Blindness Committee of the Union of Counties Association for the Blind, England. (Copies of the report are available at 1

shilling; address the Secretary, Prevention of Blindness Committee, 66 Victoria Street, S. W. London.) The proper certification of blindness and the recording of the causes of blindness have recently been stressed in Scotland, England, Wales and the United States, largely because such certification heretofore was done in a haphazard manner. The work of the clinic in Glasgow has been outstanding during the past two years in demonstrating the proper method of handling this problem. On Nov. 20, 1930, a committee of twelve members was formed to study the problem and make recommendations. In substance, the committee reported in favor of (1) examination by skilled ophthalmologists, recording (*a*) abnormalities in the visual mechanism and (*b*) the causes of such abnormalities; (2) the use of a standard form of report or certificate, and a more precise definition of blindness in terms of visual acuity and field.

CORRECTION

In the article by Drs. Jonas S. Friedenwald and H. F. Pierce, entitled, "The Circulation of the Aqueous: I. Rate of Flow," in the April issue (**7:** 538, 1932), the formula that follows the line "limits of accuracy of the chemical measurements" is part of footnote 31 and should appear at the bottom of the page.

Abstracts from Current Literature

EDITED BY DR. WILLIAM ZENTMAYER

Anatomy and Embryology

THE RETICULO-ENDOTHELIAL SYSTEM OF THE EYE. A. STILO, Ann. di ottal. e clin. ocul. **59:** 566 (June) 1931.

The author reviews recent work on the reticulo-endothelial system and the technic of intravital staining.

Trypan blue was injected subcutaneously into guinea-pigs on alternate days for sixteen days. Vital stain was not seen in the cornea or sclera, but granules of stain were present in young histiocytes in the episcleral tissues. They were also seen in the choroid and iris, in the retina near the vessels and in the conjunctiva and lids, but not in the lens, aqueous or vitreous.

S. R. GIFFORD.

Conjunctiva

OPHTHALMIA NEONATORUM. M. S. MAYOU, Brit. M. J. **2:** 973 (Nov. 28) 1931.

This report is based on the author's experience within the London area and in the work carried out at St. Margaret's Hospital. The number of cases of ophthalmia of the new-born reported in London has not diminished during the last fifteen years, and the number of admissions to the hospital has been fairly constant, but the amount of blindness has decreased by more than one-half. At present there are 218 children under 18 years of age on the roll of the London County Council blind schools. Of these, 45 (a percentage of 20.6 per cent) are blind from ophthalmia neonatorum. While this is an enormous improvement, still further improvement is possible by proper teaching and training of midwives and medical students.

The author then describes the eyes at birth and gives the histology of the conjunctiva in ophthalmia neonatorum, the results of bacteriologic examination and the method of treatment carried on at St. Margaret's Hospital.

The best irrigating solution is an acid solution of hypochlorite, 1 : 10 (eusol), but it must stand for twenty-four hours after being made. The next best is a solution of boric acid and sodium bicarbonate. After the conjunctival sac is irrigated, it is filled with acriflavine (1 : 1,500 of castor oil). Silver applications are of little value in the early stages and are apt to accentuate the symptoms unfavorably. When the swelling of the lids subsides, the silver applications still remain of the greatest value. They are applied by rubbing a 2 per cent solution into the conjunctiva with wool mops. The author is opposed to the use of silver nitrate for prophylactic treatment, and prefers acriflavine oil or protargol. External canthotomy is done in the early stages when the lids are greatly swollen.

The ordinary and detoxicated vaccines and serums have had no influence in shortening the ordinary period of the disease. Milk injections have been used without effect, and are regarded as dangerous.

The patients are kept in the hospital until there has been no discharge for a week. They are then kept under careful control to guard against relapses, of which there are about 10 per cent.

The general health of the infants is of the greatest importance, and special care must be exercised in feeding and management.

The varieties and treatment of corneal ulcer are described. In cases of corneal opacity, no operation in the way of an optical iridectomy should be done until the child is at least 6 months old. The chief indication for operation is nystagmus. The author's experience with visual benefits derived from optical iridectomy has not been gratifying. Gonorrheal arthritis, though not uncommon in mothers, is rare in babies. Rhinitis is a frequent accompaniment, and purulent mucocele is often mistaken for ophthalmia neonatorum.

This article is extremely valuable as it presents the personal experiences of one investigator who has studied every phase of this problem.

A. KNAPP.

Cornea and Sclera

HERPES AND ALLIED CONDITIONS. R. I. LLOYD, Am. J. Ophth. **14:** 601 (July) 1931.

The author discusses herpes zoster, herpes febriles and dendritic, bullous and filamentous keratitis and the rôle of trauma in allowing herpes virus access. It is believed that this same virus causes epidemic encephalitis and other nerve ailments. Disciform keratitis may be in this herpetic group. The author recommends covering the eye and the use of radiant heat in the treatment for herpes; also the thermophore, ethylhydrocupreine hydrochloride and the x-rays. Lowered corneal sensibility and various other phases of herpetic keratitis are discussed. Six cases are reported as illustrations, and a résumé of laboratory experiments with herpes virus is given.

W. S. REESE.

ARSENICAL EXFOLIATIVE KERATITIS. F. T. HYDE, Am. J. Ophth. **14:** 611 (July) 1931.

The author gives a brief history of the use of arsphenamines, and describes the onset and course of exfoliative keratitis. He remarks on the paucity of ophthalmologic literature on this condition. A white man, aged 26, with a large specific ulcer of two weeks' duration involving almost the entire sulcus of the glans was advised by the urologist to have intravenous injections of neoarsphenamine, and received 0.6 Gm. doses at intervals of about a week until six doses had been given. At the last injection the patient jumped, and a small amount of the solution was expelled into the tissues around the vein. Twenty-four hours later the arm became greatly swollen and painful, but this subsided so that he could resume work. Three days later the elbow again became swollen, and the patient complained of terrible itching. Within five days a reddish, macular rash appeared on his body, and finally variously sized blebs appeared. His eyes were red, sore and painful, and the lids much swollen. Several small areas of exfoliation appeared

on each cornea and a dirty infiltration around the right limbus. This extended until both corneas became so weakened that they ruptured. The patient was discharged practically blind. W. S. REESE.

Experimental Pathology

COMMENTS ON THE PAPER OF GOLDMAN: CRITICAL AND EXPERIMENTAL STUDIES OF THE INFRA-RED CATARACT OF RABBITS AND OF "FIRE-CATARACT." H. SCHLAEPFER, Arch. f. Ophth. **126:** 336, 1931.

This paper is the reply of Vogt and his school to the recent publications of Goldmann from Siegrist's clinic in Bern. The main difference between these two schools is that Vogt believes that the cataract produced in rabbits by exposure to infra-red light is partly due to transmission of heat from the iris and partly to direct action of the infra-red rays on the lens substance or lens epithelium, while Goldmann does not believe in the latter factor. Schlaepfer stresses the point that in all these radiation experiments the penetrating infra-red rays (shorter wavelengths) play a very important rôle irrespective of the presence or absence of the iris in the exposed eye. P. C. KRONFELD.

General Diseases

THE OCCURRENCE OF EYE LESIONS IN TUBERCULOUS PATIENTS. W. V. MOORE, Am. J. Ophth. **14:** 596 (July) 1931.

The author speaks of the infrequency of coincident pulmonary and ocular tuberculosis, and reviews the literature. Two cases of uveitis are reported in young girls with advanced pulmonary tuberculosis. The following summary is given:

This review of the literature tends to show that tuberculous eye lesions are exceedingly uncommon in patients with phthisis. On the other hand, when persons with ocular tuberculosis are examined roentgenologically, a fair proportion of them show evidences of hilus or tracheobronchial, if not of early pulmonary, tuberculosis.

Only these two cases of ocular tuberculosis were observed in six years of service as ophthalmologist to the Brooklyn Home for Consumptives. Both occurred in young women. In each case the pulmonary lesions were advanced, but there was complete recovery so far as the eye condition was concerned. During the same period not a single case of phlyctenular conjunctivitis was observed among these patients.

The consensus supports the view that, while ocular tuberculosis is probably hematogenous and secondary to a focus elsewhere in the body, most often the tracheobronchial or hilus glands, the primary lesion is almost always slight, quiescent, healed or calcified. W. S. REESE.

RELATION BETWEEN OCULAR TUBERCULOSIS AND TUBERCULOSIS OF OTHER ORGANS. TADAOKI SUDZUKI, Acta Jap. Ophth. Soc., September, 1931, p. 111.

The author has examined 490 cases of ocular tuberculosis which are divided as follows: keratoscleritis, 318; periphlebitis, 86; iridocyclitis,

67; choroiditis, 11; central retinitis, 4; neuroretinitis, 2, and conjunctivitis, 2. In these conditions the author finds that anatomically the disease is usually of an exudative or proliferative-exudative form and belongs to the second stage of Ranke's classification. Other organs of the body presented changes belonging to this second stage. Active pulmonary disease was found in very few cases and then generally in those suffering from retinal periphlebitis and iridocyclitis.

A. KNAPP.

Glaucoma

SCLEROCORNEAL TREPHINING IN CHRONIC SIMPLE GLAUCOMA. W. ZENTMAYER, Am. J. Ophth. **14**: 617 (July) 1931.

The author reports five interesting cases. In case 1, after trephining, the field remained full in one eye and showed moderate contraction in the other for ten and one-half years. One eye showed marked changes in refraction and disappearance of the glaucomatous cup after operation. Case 2 also showed marked variations in refraction after trephining. Case 3 showed an immediate progressive loss of vision following trephining as the result of low grade iridocyclitis, which resulted in marked deposition of pigment over the tissues of the anterior chamber. Case 4 also showed pigment migration following trephining. In case 5 the patient lost the right eye through late infection, and showed gradual loss of vision in the left over a period of six years, both following a trephining. As a result of the aforementioned experiences, the author resorts to this operation less frequently than formerly.

W. S. REESE.

SOME POINTS IN CONNECTION WITH THE TREATMENT OF GLAUCOMA. R. H. ELLIOT, Brit. M. J. **2**: 1169 (Dec. 26) 1931.

Colonel Elliot believes that the treatment of glaucoma today is a compromise of operative and medical treatment. He discusses medical treatment under the following headings:

Miotics: Pilocarpine is preferable to physostigmine (eserine) in most cases because it causes less irritation and less tendency to spasm and headache, and when suitably employed is as effective as physostigmine. Pilocarpine should be used in the weakest dose that will maintain miosis, and should be given the last thing at night, the first thing in the morning and at about 3 o'clock in the afternoon.

Massage: Massage is not sufficiently appreciated. The technic is simple, and any patient can be taught to use it.

General Rules: To influence or to prevent vasomotor exhaustion, the patient should be advised to avoid overfatigue, to get regular sleep and meals, to use very little alcohol and to avoid stooping and sources of strain, particularly avoiding all causes of anxiety. The general health of the patient should be carefully noted, and the general standard of health should be improved in every possible way.

If this regular treatment is faithfully carried out, the patient's eyes from time to time have to be carefully examined to determine whether the condition is stationary. The author then draws attention to the

various factors that are of importance in examination of the eyes, such as tension, curtailment of the fields and cupping of the disks.

As soon as the surgeon has satisfied himself that nonoperative treatment has failed to hold the disease in check, operation should not be delayed.

The prognosis is apt to be influenced by the stage at which the operation is undertaken. While the risk in ordinary and straightforward cases need not be exaggerated, the outcome of the advanced cases is not assured, and the patient or his relatives should be acquainted with the situation.

In diseases that occur in people advanced in years the expectation of life is an important factor when decision concerning an operation is made, although very old patients often stand trephining very well.

A. KNAPP.

HISTOLOGIC FINDING IN GLAUCOMA AND HOMOLATERAL NEVUS FLAMMEUS OF THE FACE. WALTER JAHNKE, Ztschr. f. Augenh. **74:** 165 (May) 1931.

The association of nevus flammeus and glaucoma on the same side of the head has been discussed on many occasions, but only six cases (seven with the author's) have been described histologically. According to the author, most of the cases reviewed were congenital hydrophthalmos and the others appeared in later life. Even though no definite history was found concerning the involvement of the eye in early childhood, the author believes that hemangiomas of the choroid, presumably like most angiomas of the face, are congenital.

The histologic picture presented in some of the cases showed an occasional obliteration of the canal of Schlemm and occlusion of the iris angle as the possible cause of glaucoma. Jahnke considers the increased tension as due to the inability of fiuid to exit through the canal of Schlemm, and he believes that the nevus and hydrophthalmos are anomalies which are present because of a disturbance of the germ layer.

A. M. YUDKIN.

Hygiene, Sociology, Education and History

THE EYESIGHT OF MOTOR DRIVERS. N. BISHOP HARMAN, Brit. M. J. **2:** 854 (Nov. 7) 1931.

The accidents known to be due to poor sight are rare. The standards of vision vary in different countries. Some are very severe, as in Germany. It is generally regarded that for safety sight should have a threefold quality: visual acuity, field of vision and binocular vision. Without good or sufficient field of vision, the fullest visual acuity is useless. The need for binocular vision is a more debatable point; the author is convinced that it makes for a good and safe driver. He does not like the one-eyed driver, and believes that much of the difficulty drivers experience in reversing is due to the fact that they are driving with one eye.

As night driving is becoming more common the light sense becomes of special importance. It is common to find that myopic persons dislike night driving, as from the form of their lenses the light entering their eyes is reduced. To determine whether a doubtful light sense is safe or not, the author suggests mapping out the peripheric field with the perimeter in daylight and then reducing the illumination to one-tenth by adjusting the window blinds. The excessive reduction of the field of vision in those with defective light sense will then be manifested.

Defects of color should not constitute any disability for driving either during the day or night, though the growing use of colored signal lamps at road crossings may present a real difficulty. The author feels that a much more simple and safe signal should depend on form rather than color, and suggests a horizontal and vertical bar of light.

An important factor in this problem is the interrelation of sight and muscular coordination of the driver. Many accidents are due to defective reaction time. Visual anesthesia, or wandering attention, has caused many deaths. The conditions which unfavorably affect this reaction time should therefore be avoided, and it may be necessary to have a double standard of fitness for drivers according to the kind and time of driving.

A. KNAPP.

Lens

DO THE ULTRAVOLET RAYS ASSIST IN THE DEVELOPMENT OF GLASS-BLOWER'S CATARACT? A. VOGT, Klin. Monatsbl. f. Augenh. **86:** 295 (March) 1931.

The author denies the claims of some recent publications which state that ultraviolet rays take part in the development of glass-blower's cataract. Conceding that ultraviolet rays of certain alkalis and rare earths emit special, damaging rays at a temperature of 1,500 degrees, Vogt states that the traces of such rays could not be detected in the heat of blazing glass-blower's furnaces. Ultraviolet rays cannot take any part in the development of cataracts produced experimentally by infrared rays, for the ultraviolet has been completely eliminated by means of a filter containing compound solution of iodine.

Photographic copying paper, made especially sensitive to ultraviolet rays, and exposed to daylight at noon, even during winter, no matter whether the sky is clouded or not, will completely blacken after a certain number of minutes. The same kind of paper, placed in front of a glass-worker's furnace, will require more than a hundred times the daylight exposure to blacken equally. The paper will still be completely unchanged after a three or more hours' exposure to the blazing glow of the glass-blower's furnace. This experiment shows that the usual, diffused daylight contains vastly larger quantities of ultraviolet rays than the heat of a glass furnace. It also proves that the glass-blowers would be more secure against a cataract at his furnace than in the broad daylight, if the ultraviolet rays were the damaging factor, as has been claimed. Vogt feels certain that the erroneous idea that glass-worker's cataract is caused by ultraviolet rays would disappear from ophthalmologic literature if future writers on this topic would repeat his simple experiment.

K. L. STOLL.

Methods of Examination

MIRROR FOR INSPECTION OF THE FORNIX. GEZA MUSITZ, Klin. Monatsbl. f. Augenh. **86:** 513 (April) 1931.

Realizing the difficulty of examining the fornices of the conjunctival sac, the author constructed a small metal mirror, which is blunt-edged, oval and curved to fit the convexity of the globe. It measures 20 by 12 mm., is highly polished and is covered with a bright silver coating. A handle is attached at an angle of 45 degrees. To insert the mirror, the lids are lifted off the globe, Desmarres' lid elevator being used. One of the extremities of the mirror, measuring 12 mm., is sufficient for inspection in persons with a narrow palpebral fissure or in children.

K. L. STOLL.

A NEW METHOD OF LOCALIZING FOREIGN BODIES IN THE EYE: TELERADIOGRAPHY WITH VISUALIZATION OF THE CORNEA. HUGO AHLBOM, Acta ophth. **9:** 1 1931.

The author presents a brief summary of the more important methods described in the literature for roentgenographic localization of foreign bodies in the eye. An account of a new method is given. It is based on the taking of the two images in two places at right angles to each other, one of them being a profile picture with the cornea visible. Both pictures are taken with a focus plate distance of 2.75 meters. The cornea is rendered visible in the lateral view by placing an aluminum wedge between the patient's eye and a cassette. The patient is seated during the examination, and special measures are adopted to enable the investigator to control the direction of the line of vision during exposure. As an extra safety measure the principle of the so-called physiologic method, i. e., change of the directions of vision, is applied. The localization is carried out directly on wet films with the aid of patterns, and the interpretation is made on transparent film by employing direct measurement with a pair of compasses. No contact markers are necessary for this localization.

A. M. YUDKIN.

OCULAR COMPLICATIONS OF CHRONIC EPIDEMIC ENCEPHALITIS. T. H. WHITTINGTON, Brit. M. J. **2:** 981 (Nov. 28) 1931.

The author has examined 174 cases in the later stages of encephalitis in children and has reviewed the subject from an ophthalmologic standpoint. He points out that encephalitis is of interest to ophthalmologists for the following reasons:

1. The ocular changes are of great importance in the diagnosis.

2. These ocular signs are peculiar and are some of the most extraordinary that occur in the disease.

3. The pathology of the disease throws some light on the neurology of the ocular movements and particularly on ocular palsies.

4. As it is now recognized that great benefit is obtained in chronic encephalitic parkinsonism by the use of the belladonna group, the resulting visual changes may have to be counteracted by the ophthalmologist.

In the more acute stage there are transient nerve root paralyses, deeper lesions resulting in altered movements of the eyeballs, defects in

pupillary reactions and peculiarities in movements of the lids. In the Ministry of Health Report, no. 11, in 78 per cent of the patients the external ocular muscles were affected; there was ptosis in 50 per cent, strabismus in 34 per cent, diplopia without obvious squint in 18 per cent and nystagmus in 18 per cent.

There are two common types in the acute stage of epidemic encephalitis with which the ophthalmologist may meet: The first is a type showing ocular disturbances occurring in a febrile disease with marked disorders of sleep. The second is that of a so-called influenza with diplopia.

The presence of papilledema or optic neuritis is decidedly against the diagnosis of encephalitis. While the ocular signs and symptoms are so closely present at the onset and during the acute stage, they are comparatively rare in the postencephalitic stage.

The author gives statistics in 174 cases. The points to be noted are: (1) the infrequency of ptosis; (2) the number of cases with disturbed pupillary reactions; (3) the absence of paralytic squint, but the remarkable number with concomitant external strabismus or some form of divergence (28 per cent), and (4) the onset of oculogyric crises.

Ptosis seems to be the result of a lymphocytic inflammation in the acute stage, which affects the third nerve as it passes out from the surface of the crus.

The pupillary changes are due to a lesion of the third nerve nucleus or its connections.

The author suggests that the squint found in these children may be explained by a disease of the higher control of the lower centers in the midbrain.

The oculogyric crises are peculiar to parkinsonism. There generally is a spasmodic movement of the eyes upward, common slightly to one or two sides, lasting from one minute to many hours. These crises come on from one to eight years after the original acute illness. Periodic outbursts of uncontrolled behavior may be associated with these crises in adolescence. The midbrain, particularly the substantia nigra and corpus striatum, shows most signs of the disease, and so the connecting links for central control over the extrapyramidal system are affected. There seems to be a coordinated upward movement of the eyes associated with the emotions and related to a center at a lower level.

Other ocular symptoms are excessive blinking, fluttering of the eyelids and difficulty in moving the eyes in a vertical direction. The author believes them to be rather characteristic of the overemotional state of patients than typical of chronic encephalitis.

Scopolamine or stramonium is given in the treatment for parkinsonism; often ten times the usual dose is given, resulting in marked improvement of the parkinsonian rigidity. Many patients complain of their inability to read and of being worried by bright light. This loss of accommodation can be relieved with physostigmine (eserine) or pilocarpine.

This is an extremely interesting study of the important changes that are now so prevalent. It deserves careful study in the original.

A. KNAPP.

Neurology

OBSERVATIONS ON OCULAR PALSIES. GORDON HOLMES, Brit. M. J. **2:** 1165 (Dec. 26) 1931.

Ocular palsies may be divided into disturbances of the upper motor neurons, which carry impulses from the higher centers to the motor nuclei and their nerves, and the lower motor neurons, which are the result of lesions of the nuclei and the nerves that innervate the muscles directly. In lesions of the upper motor neurons, movements as such are lost and sometimes those that normally are excited by certain impulses only are also lost, while in diseases of the lower motor neurons, the muscles themselves are paralyzed.

Two types of supranuclear palsy can be distinguished: the so-called associated paralysis and the true supranuclear palsies, in which movements of the eyes in certain directions are lost to certain stimuli only.

The author divides ocular palsies into three main groups: (1) paralysis of the ocular muscles due to disease of their motor nuclear nerves or nuclei, (2) conjugate palsies in which the movements of both eyes in one or more directions are lost to all stimuli and (3) supranuclear palsies in which movements of the eyes can be excited by certain stimuli only.

According to the anatomic site of the lesion, ocular palsies may be divided into: (*a*) nuclear palsies in which the nucleus is involved, (*b*) stem palsies due to interruption of motor fibers as they pass through the brain stem and (*c*) root palsies in which the extramedullary portions of the nerves are affected.

The author then takes up in detail nuclear palsies, stem palsies, root palsies, conjugate ocular palsies and supranuclear ocular palsies.

This lecture is so admirably written and contains so many important observations in a short space that an abstract is not possible; the reader is urged to study the original communication.

A. KNAPP.

A CASE OF PARALYSIS OF THE LATERAL MOVEMENTS OF THE EYES FROM A PONTILE LESION: A CONTRIBUTION TO THE STUDY OF OCULOGYRIC PATHWAYS. P. VAN GEHUCHTEN, Rev. d'oto-neuro-opht. **8:** 701 (Nov.) 1930.

Stimulated by the suggestion of Andre-Thomas that a large number of careful postmortem observations in cases of associated paralyses of the ocular muscles are necessary in order to enhance knowledge of the mechanism involved, van Gehuchten records the complete history in a case, with detailed autopsy observations and serial microscopic sections of the brain stem. This is followed by a discussion of the known facts about the pathways and by a comparison of the lesions in his case with these facts.

The patient, aged 30, suffered from phthisis. The ocular symptoms began with diplopia, due to paralysis of the left external rectus muscle, which was soon followed by conjugate deviation of the eyes to the right

and paralysis of the left facial nerve. Convergence was good. On looking to the right, an intense nystagmus to that side was noted. Later there were hyperesthesia in the domain of the right trigeminus nerve, hypo-excitability of the left labyrinth and a slight pyramidal lesion situated in the left side of the bulb near the floor of the fourth ventricle at the level of the knee of the facial nerve. Six months later the ocular paralysis was unchanged, the spontaneous nystagmus had disappeared, the facial paralysis was accentuated, and there were a slight diminution of hearing in the left ear, paralysis of the recurrent nerve, little reaction to stimulation of the left vestibular apparatus and paralysis of the left trigeminus nerve.

Autopsy revealed a large whitish tumor (tuberculoma) in the bulb and pons situated to the left of the median line and extending from the middle of the bulbar olive to the upper limits of the pons.

A discussion of the relation of the microscopic observations to the symptoms and their bearing on the oculogyric pathways follows.

W. ZENTMAYER.

Ocular Muscles

SUPRANUCLEAR PARESIS OF THE INTERNUS RECTUS MUSCLE AND INTERNUCLEAR OPHTHALMOPLEGIA. P. A. JAENSCH, Arch. f. Ophth. **125:** 592, 1931.

The author has collected all of the cases of supranuclear lesions of the lateral motors that were seen in the clinic of Professor Bielschowsky (Breslau) during the course of the last few years (twenty cases). He records three cases in which there was a paresis of one or both recti interni on lateral rotation, while the same muscles responded normally on convergence. More common are cases in which the supranuclear paresis of the internus rectus is associated with a paresis of one abducens (internuclear ophthalmoplegia). The lesion in these cases lies in or around the posterior longitudinal fasciculus. If the pathologic process lies closer to the nucleus of the third nerve (internuclear anterior ophthalmoplegia), then the rectus internus is more affected than the corresponding abducens. In the opposite case (a lesion in the caudal portion of the posterior longitudinal fasciculus, internuclear posterior ophthalmoplegia), the rectus externus is more paretic than the synergistic internus. The etiology was epidemic encephalitis in eight cases and multiple sclerosis in five cases. Syphilis apparently does not produce this type of lesion.

P. C. KRONFELD.

PARALYSIS OF ACCOMMODATION AND DISTURBANCES OF THE PUPILS AFTER LEAD POISONING: REPORT OF A CASE. N. BLATT, Klin. Monatsbl. f. Augenh. **86:** 482 (April) 1931.

Statistics show that lead poisoning, on which a great number of publications have appeared for over a hundred years, has been reduced about 50 per cent since 1900. In 320 papers on changes caused by lead poisoning in every tissue of the eye, Blatt found references to changes in the pupils in only 21, and to disturbances of accommodation in only 3.

The case reported by Blatt himself concerns a man, aged 37, who was employed as a plumber and welder. He suffered in 1927 from a sudden attack of vertigo, colic, vomiting, obscurations and transient blindness. These symptoms disappeared after a resting period. Two years later he experienced a sudden sensation of heat in his head and body, accompanied by obscurations of vision, scintillations and inability to write and read. These symptoms persisted for about two weeks, appearing repeatedly every day, synchronous with attacks of colic. During these attacks decided anemia of the retina was observed. There was a lead seam on the gingiva, but no pathologic symptoms. The blood pressure, blood count and vision were normal. The pupils were equal, measuring 5.5 mm. in diameter; their reaction to light and in accommodation was sluggish; their shape was distorted to an obtuse triangle. During the attacks of obscuration the pupils became from 1 to 2 mm. larger and fairly round, with hardly any reaction. The near point of both eyes was at 56 cm. instead of at 20.2 cm., according to the patient's age; the latitude of accommodation was 1.7 convex diopters, or 3.35 diopters less than normal. The patient could read the finest print of Snellen's type at 30 cm. only with 2.5 convex diopters. Rest in bed and iodines internally brought a decided general improvement in about a month; the obscurations became less severe and less frequent, the pupils contracted to 4.5 mm. and showed a more prompt reaction, and the near point had approached 48 cm. He returned to work a month later, but changed his occupation. No general or ocular symptoms remained, and vision stayed perfect from that time until September, 1930, when he was last examined.

The author agrees with Elschnig that the photopsias, obscurations and attacks of temporary blindness in lead poisoning are caused by spasms of the retinal vessels. He wishes the term "lead amaurosis" reserved for this condition solely. Lead may produce the same changes in the vessels as arteriosclerosis. Spasms are found in the vessels of the brain, kidneys and intestines. Obscurations due to disturbance of the retinal blood circulation are transient. Endarteritis, however, has been observed in the retinal vessels. Gradually developing blindness is due to pathologic changes in the fundus, especially in the optic nerve, and is permanent. This sudden transient and the slowly developing types of amaurosis in lead poisoning are clearly described by Wilbrand-Saenger. Blatt recorded 22 cases in an appended table, 7 of which showed absolute and 6 nearly absolute rigidity of the pupils; mydriasis was present in 12, and miosis in only 3 of these 22 cases; 3 of the patients had Argyll Robertson pupils, 2 binocular and 1 monocular; 6 had pupils of normal size. The shape of the pupils was normal in all but 3 cases. The size of the pupils is important, according to Wilbrand-Saenger, who localized the focus beyond the primary optic centers in all cases with intact pupils. The changes of the pupils and the anomalies of accommodation may be caused either by direct toxic action of the lead poison on the tissue of the nerves or indirectly by hyaline degeneration, endarteritis or perivasculitis of the blood vessels and the resulting inhibition of function. The cessation of the disturbances after a change of occupation seems to point to the first possibility, while permanent changes are due to lesions in the nerve centers brought on by vascular pathologic changes.

K. L. STOLL.

Operations

SENILE CATARACT EXTRACTION IN THE CAPSULE. A. ELSCHNIG, Ztschr. f. Augenh. **75:** 1 (Aug.) 1931.

Elschnig recommends the following procedure for senile cataract extraction in the capsule. Two days before the operation, the lacrimal tract, lids and culdesac are irrigated with hydrargyrum oxycyanatum, 1 : 5,000, and a modified yellow mercuric oxide salve is applied. When no bacteria are present on two consecutive days, the operation is performed. The muscles of the lids are immobilized by an injection of a 2 per cent solution of procaine hydrochloride-epinephrine into the layer of the panniculus adiposus in the area of the outer half of the zygomatic arch. A stay suture in the superior rectus tendon is employed to steady the movements of the eye.

After several instillations of 3 per cent solution of cocaine, 1 cm. of a 2 per cent solution of procaine hydrochloride-epinephrine is injected into the orbit by a needle pierced through the skin in the outer, lower portion of the orbital socket. Only in very sensitive patients is the retrobulbar injection fortified by a subconjunctival injection of 2 per cent solution of cocaine into the upper equatorial region of the eyeball. All subconjunctival injections are made without fixing the tissue with forceps. Care is taken not to make the injection into the superior rectus tendon. The incision is made through the upper two fifths of the arc in the cornea of normal size, but in smaller corneas it is made larger. Sutures are placed in the conjunctival flap before the corneal incision is made. The conjunctival flap is then lifted with forceps, the anterior chamber is opened, and the iris is slightly cut, close to its root, with deWecker scissors. With specially made forceps the capsule of the lens is grasped for about 1.5 mm. in the lowest part of the dilated pupil. The lens is moved gently from side to side for about eight or ten seconds. The lens is then lifted with the forceps, while the lower margin of the lens is pushed upward so that the lens tumbles and its lower edge engages the wound. It is imperative for the lens to come through the round pupil in order that the iris will not be injured. The wound is closed with the prepared suture, and the iris is reposited. Physostigmine (eserine) is instilled. The wound is gently touched with a 5 per cent tincture of iodine. Physostigmine oxycyanide salve is then applied to the area operated on, and the eyes are bandaged.

The eye, as a rule, shows no disturbance during the patient's stay of from nine to ten days in the hospital. The eye is bandaged daily. Atropine is rarely necessary. A drop of cocaine is instilled daily. If irritation is present a 1 per cent solution of homatropine is instilled. The suture is left in for one week.

The author performs iridectomy for old people (over 78), for adipose persons, for patients with emphysema and for patients in whom an iris prolapse might be expected. In cases of high myopia, iridectomy is not recommended. A canthotomy, as suggested by Axenfeld, in addition to an immobilization of the muscle of the lid, is preferred.

Elschnig finds that in composed patients the Stanculeanu-Knapp-Torök Elschnig intracapsular operation is practicable in 80 per cent of

all cases of cataract. It is safest in cases of immature nuclear or posterior cortical cataract. This procedure is not adaptable to cataracts that show a tendency to swelling (intumescent cataracts).

The visual acuity in the eyes operated on by the aforementioned method is usually good. If the suture is properly placed, the astigmatism produced by the operation is no greater than that obtained by any other method.

A. M. YUDKIN.

Physiology

THE CHEMICAL CONSTITUENTS OF THE AQUEOUS, VITREOUS AND LENS. C. S. O'BRIEN and P. W. SALIT, Am. J. Ophth. **14:** 582 (July) 1931.

This is a technical article that does not lend itself to abstracting. A summary is given of the different constituents of aqueous, vitreous and lens, and their percentages as found by the authors.

W. S. REESE.

THE CONVERGENCE OF HUMAN BINOCULAR VISION. N. A. STUTTERHEIM, Brit. J. Ophth. **16:** 20 (Jan.) 1932.

Fusion of the two ocular images is a psychologic phenomenon. There is no actual fusion either in an optical or a physiologic sense.

In every squint one of the ocular axes necessarily deviates from the object of vision; consequently every squint is divergence. In ophthalmology a position of convergence does not occur. A position of divergence is every position in which the two visual axes do not meet an object of vision, e. g., the natural position of binocular rest, in sleep, in deep narcosis, probably even in staring.

Convergence is a visual movement, and divergence never is. Every visual movement of the human binocular organ is convergence. This convergence is the obligatory factor of every visual binocular movement. It is therefore more than a function; it is the kinetic principle of binocular vision.

Testing of convergence is made at 5 or 6 meters. The object of vision is a narrow white strip on a black background. Loose prisms are placed before the eyes until the images are no longer merged. The preceding number of prism degrees was taken as the result of the test. When the eyes are looking at a distant object straight ahead, it is called the functional primary direction. It is immaterial whether the prism is placed with its base out or in, up or down, or in whatever combination of these positions, the dual eye which overcomes the difficulty of the deviation and performs single vision, performs this by the power of convergence.

This new conception of convergence affects the diagnosis of asthenopia. It displaces the center of gravity in that diagnosis from the static factor of monocular refraction in the direction of the kinetic factor of functional binocular ability. An important physiologic difference was found in this function between the Bantu and the European.

W. ZENTMAYER.

THE CONTENT OF SOLUBLE PHOSPHORUS IN THE AQUEOUS. A. STILO, Ann. di ottal. e clin. ocul. **59:** 718 (Aug.) 1931.

From 10 to 15 cc. of aqueous from the eyes of animals (species not given) was tested according to the method of Longo. The phosphorus recovered was chiefly in inorganic form, including traces of pyrophosphates and larger traces of complex ethers. These are the same forms as those found in the blood plasma but in much weaker concentration, and their presence suggests the origin of the aqueous as a dialysate of the blood.

S. R. GIFFORD.

THE EXPERIMENTAL FOUNDATION OF BINOCULAR COLOR VISION. ERNST FLEISCHER, Arch. f. d. ges. Physiol. **228:** 724, 1931.

Charts are described for use in the stereoscope on which two colorless rectangles are pictured on a colorless background. The two rectangles are of different shades. Series of such charts were made ranging from a black background to a white background. When these were examined in the stereoscope and the two rectangles fused binocularly, the following occurred:

When the two rectangles were both lighter than the surrounding field, the lightest one was suppressed and the darker commanded the field. When the rectangles were darker than the surrounding field, the lighter commanded the attention. When, however, one rectangle was darker and the other lighter than the surrounding field, then rivalry occurred, and first one and then the other was perceived.

Similar charts were viewed through colored filters, and the results interpreted in terms of the three component theory of color vision.

F. H. ADLER.

Refraction and Accommodation

SCLEROKERATOMETER FOR FITTING CONTACT GLASSES. R. HELMBOLD, Klin. Monatsbl. f. Augenh. **86:** 510 (April) 1931.

In order to obtain properly fitting contact glasses, exact measurements of the curvature of the anterior portion of the globe are necessary. The ophthalmometers of Helmholz, Javal-Schioetz and others being insufficient for this purpose. Czapody took a cast of the cornea and sclera, using paraffin of a low melting-point, from which the contact glass is made. Several technical considerations render this method fallible. Helmbold, therefore, constructed an apparatus, consisting of a container with forty-two metal rods, placed in a space of 20 mm. These rods are arranged so that they can be moved parallel to one another and then be fixed by a screw device in any desired position. The ends of the rods form a narrow band which adapts itself to any curvature, the profile of which is recorded with a pencil on a piece of paper. Measurements are taken in several meridians, especial care being taken to record the corneoscleral sulcus and the adjoining radius of the sclera. Good results have been obtained and no damage was done to the eyes, but it is necessary to ascertain whether the weight of the apparatus will slightly change the radius of the cornea and sclera during examination. Disks to test the sclerokeratometer prior to each examination have been devised by Dr. Hartlinger of C. Zeiss & Company.

K. L. STOLL.

Retina and Optic Nerve

RETINITIS PSEUDO-NEPHRITICA STELLATA. E. PUSCARIU and J. NITZULESCU, Brit. J. Ophth. **15**: 697 (Dec.) 1931.

The authors have adopted the opinion that so-called retinitis albuminurica should be called the neuroretinitis of Bright's disease, in order to point out first the constancy with which the papillary lesions are found and second that there is not such a causal relationship between retinitis and nephritis as the usual name would imply. In fact, Bright's disease seems to be not a simple process in the kidneys but a more general metabolic disturbance touching at the same time, independently, the kidney, the retina and other territories in the body.

The authors state that the most authoritative workers, however, tend today to admit the direct, immediate influence of local circulatory troubles on the production of retinal lesions, and that such troubles are due to some toxic influence of renal or general origin.

Three cases of pseudonephritic stellate retinitis are reported. From a study of these, together with the literature, the authors submit the following summary:

"Retinitis pseudo-albuminurica stellata (among which we class our three cases given in this paper) occupies an intermediary position, as a transition between the typical Brightic forms and the different exudative ones, as described by Junius and Horniker. Their study gives us a better understanding of the pathogenesis of Bright's neuroretinitis, suggesting to us a similar mechanism in the production of the lesions.

"The different aetiological factors acting on the retina (acute or chronic infectious diseases, cranial traumata, cerebral tumours, serous meningitis) determine (probably through a vascular mechanism) their related pictures, which represent somewhat the special kind of reaction of the retina, and differ in details according to the different cases and aetiologies. At the origin of the pseudo-Brightic retinitis, due to intracranial tumour, there seems to be a mechanical lymph stasis, causing the papillary oedema and through it metabolic troubles. At the origin of the pseudo-Brightic, infections, etc., retinitis the local troubles seem under the dependence of general metabolic processes. All follow thereafter a similar evolution and produce related fundus pictures.

"The neuroretinitis of nephritics is not connected by any causal link with the kidney lesions and therefore the only justified name should be that of Bright's neuroretinitis (understanding Brightism as a general disease, with renal, ocular and general manifestations).

"From the facts discussed here, we incline to admit that the production of the neuroretinitis is independent of arterial hypertension, which, like the renal lesions, is only a symptom in evolution under the influence of the same causes.

"The difference in evolution and prognosis between the Brightic forms and the so-called pseudonephritic is due to their different aetiology and to the intensity with which this affects the general state. This explains the benignity of those due to local processes and on the contrary the gravity of those where the whole organism is participating (hypertension, Brightism).

"The characteristic clinical picture of Bright's neuroretinitis shows that the special metabolic trouble, which forms its basis, has in its nature intensity and distribution of vascular action, particular and constant characteristics."

W. ZENTMAYER.

GONIN'S OPERATION FOR RETINAL DETACHMENT. G. NICOLETTI, Ann. di ottal. e clin. ocul. **59:** 636 (July) 1931.

The author reviews previous work and gives a detailed account of Gonin's theory of pathogenesis, method of localizing and technic of operation. The method, as observed by the author in the clinic at Padua, has given results which proclaim it as the method of choice. No case reports are given.

S. R. GIFFORD.

THE SYMPTOMATOLOGY OF RHINOGENIC RETROBULBAR NEURITIS. G. SANNA, Ann. di ottal. e clin. ocul. **59:** 730 (Aug.) 1931.

Ten case histories are presented. Most cases were not seen until permanent changes in the nerve had occurred, and the evidence in favor of a rhinogenic origin consisted chiefly in the presence of roentgenologic signs of definite sphenoiditis or ethmoiditis on the corresponding side. Varying types of field defect were present, and cases with chorioretinal changes were included. No results of treatment directed to the sinuses are reported, so that the author's conclusions with regard to the group of cases may be considered doubtful.

S. R. GIFFORD.

HOLE AT THE MACULA. JOSE A. SEÑÁ, Arch. de oftal. de Buenos Aires **6:** 554 (Sept.) 1931.

This is the report of a case with all the characteristics of a lesion of traumatic origin but in which the absence of all traumatic history and of other etiologic factors leads the author to consider it as spontaneous, any etiopathogenic theory of its production being purely hypothetic. The report is preceded by a detailed study of the histology of the macula, of different pathologic processes here situated and of the history and present status of our knowledge in connection with this lesion.

C. E. FINLAY.

DETACHMENT OF THE RETINA FOLLOWING DETACHMENT OF THE CHOROID. H. H. ELSCHNIG, Klin. Monatsbl. f. Augenh. **86:** 595 (May) 1931.

A week prior to Elschnig's examination a man, aged 42, suffered from a disturbance of the vision of his left eye and retinal hemorrhages, which were observed by another oculist. Elschnig found in the lower nasal portion of the fundus a hemispheric prominence of the retina, which was well defined backward, where another flat prominence, about 2 disk diameters in size, was seen extending close to the disk. The region of the ora serrata was involved. No shadow could be discovered by diascleral transillumination; the detachment was rigid and presented the same red hue as the surrounding fundus. The number of prominences increased during the following days, so that the char-

acteristic design of the ora serrata and the ciliary body could be distinguished on the nasal inferior prominence. In the expectation of finding a subchoroidal cyst, or a cysticercus, a fenestrated opening, 2 mm. wide and 5 mm. long, was made in the sclera at the site of the largest prominence, about 6 mm. behind the limbus, whereupon a clear, stringy fluid escaped. The opening was closed by sutures; recovery followed promptly, and vision was 6/8 two months later. The lower portion of the retina, especially in its periphery, showed scattered pigment spots.

Elschnig compares this primary detachment of the choroid with those found in the most anterior portion of the fundus after intracapsular extractions, although secondary detachment of the retina has not been observed after that of the choroid. The etiology of the subchoroidal fluid could not be explained.

K. L. STOLL.

Trachoma

CLASSIFICATION OF TRACHOMA. SZYMANSKI, Rev. internat. du trachome **8**: 117 (July) 1931.

While many classifications of trachoma exist, the better known being those of MacCallan, Cuenod and Nataf, and Majewski, it is the author's desire to submit a more practical type which contains all the clinical forms, the various stages and the anatomicopathologic periods and variations. His classification verbatim is as follows:

	I. Stadium Incipiens
A. Forma humida	1. Trachoma acutum mixtum
B. Forma sicca	2. Trachoma latentum circumscriptum
	II. Stadium Evolutum (Floridum)
A. Forma humida	3. Trachoma granulosum, generalisatum, disseminatum
	4. Trachoma molle bulbare
	5. Trachoma papillare diffusem
B. Forma sicca	6. Trachoma gelatinosum
	7. Trachoma induratum (tarsitis)
	8. Hemiptosis trachomatosa
C. Forma complicata	9. Trachoma cum pano
	10. Trachoma cum ulcere corneae
	11. Trachoma cum trichiasis et distichiasis
	III. Stadium Terminale Degenerativum
A. Forma sicca	12. Trachoma cicatricum
	13. Trachoma xeroticum
B. Forma complicata	14. Trachoma cum entropione
	15. Trachoma cum eversione palp. inf. et stenosi punctilacrimalis
	16. Trachoma cum symplepharo
	17. Trachoma cum ankyloblepharo
	18. Trachoma cum leucomate
	19. Trachoma cum staphylomate
	20. Amaurosis binocularis trachomatosa

The cicatricial period is divided into regressive and degenerative by MacCallan. The author believes this division to be superfluous. The incubation period which exists for one or two days is not included because of its difficulty of establishment.

The author stresses the differentiation of the dry and wet types, and cites as his reasons for using it in the classification, the contagiousness of the wet type and the different treatment necessary.

L. L. MAYER.

TRACHOMA IN BELGIUM AND THE WORK OF FOREIGNERS. R. HUBIN and J. HUBERT, Rev. internat. du trachome **8:** 126 (July) 1931.

In 1928, Van Duyse, in his report concerning trachoma in Belgium, was very optimistic and brought evidence to bear on the fact that the condition was not spreading. The authors show figures that would indicate an increase, especially in Liége. For the most part the cases are contaminations from foreign workers. A difference in the type of transmitted trachoma is noted in those coming from Algeria, Morocco and Italy, where the torpid or dry type is evidenced, while from the eastern countries, Serbia, Hungary and Russia, a florid, acutely symptomatic type is found. In spite of the fact that clinics in other Belgian localities show no increase or little evidence of finding new cases, the authors are imperative in their demand that a close watch must be maintained by the hygienic department in order to forestall dissemination of the disease.

L. L. MAYER.

THE INTRADERMAL REACTION IN TRACHOMA. R. TRICOIRE, Rev. internat. du trachome **8:** 144 (July) 1931.

In 1923 the author proposed a diagnostic intradermal test for trachoma. Twenty-five hundredths cubic centimeter of the preparation is used. It is prepared as follows: Trachomatous granules, preferably from an untreated subject, are mixed aseptically with ten times their volume of physiologic solution of sodium chloride, triturated with a glass rod and allowed to macerate in an oven at 37 C. After two weeks in the oven, it is centrifugated in sterile tubes; the liquid that is on top is removed and added to its volume of physiologic solution of sodium chloride. This is the reagent. To prevent any chance of infection, 2 drops of tincture of iodine is added to each 10 cc. of the reagent. The iodine may be neutralized after one-half hour with a drop of sterile sodium hyposulphite. A positive reaction is noted in the appearance of a red, painless papule, which appears about forty-eight hours after injection and may persist for two or three days. In nontrachomatous cases, there is no reaction. The results obtained by various workers are cited. R. C. Mikaélian found the reaction positive in 68 per cent of his cases of trachoma. Where the Porwaczek bodies were found, the reaction was usually positive. R. Bellot found 70 per cent positive reactions. Most authorities agree that positive reactions are obtained in the florid type, while in older cases in which cicatrix has been formed the reactions are doubtful or negative.

The author believes that the greatest value of the test is in estimating the contagiousness of trachoma.

L. L. MAYER.

INCIPIENT TRACHOMA: CICATRIZATION AND RELAPSES; THEIR DIAGNOSTIC VALUE. E. PUSCARIU, Rev. internat. du trachome **8:** 159 (July) 1931.

The progress of late in the study of trachoma has been mostly in bacteriology and biology, and the author hopes for further study of the local lesions. Relapses in incipient trachoma are rare. Four cases are reported. All of the patients were discharged after treatment as cured, with no evident follicles. They returned later with scarring due to treatment and new follicles. There may be some reason to think of a secondary infection or a reinfection with trachoma in these cases, but the author thinks that the virus simply remained dormant over a period of time.

L. L. MAYER.

Tumors

RETINAL GLIOMATA TREATED BY RADON SEEDS. R. F. MOORE, H. B. STALLARD and J. G. MILNER, Brit. J. Ophth. **15:** 673 (Dec.) 1931.

Five cases are reported in detail to show a means of treating a condition for which it can be said there has been no alternative but removal of the eye, and when, as is frequently the case, both eyes are affected, the only remedy available in the past has been the removal of both eyes, a mutilation to which parents will often refuse to submit their children. From 1871 to the end of 1924, there have been reported at Moorefield's Eye Hospital 163 cases of glioma of the retina, and of these the proportion of bilateral to unilateral was 1 : 5.11.

Described in detail are the clinical features of the five cases, the technic, the choice of case, the changes which were observed to take place in the growths and tissues of the eye and the results obtained. In two only of the five cases was it possible to watch, for any considerable period, the disappearances of the growths and the subsequent changes which took place in them.

The author's conclusions are as follows: It is evident that the data that are here presented are too scanty for much that can be properly set under the head of conclusions; however, sufficient has been shown to guide us in further investigations, and enough it may be thought to justify further treatment along these lines, more especially in bilateral growths.

It is demonstrated that a seed of at least 5.3 millicuries may be inserted into the eye, and that at least five seeds, each of a strength of 3.5 millicuries, may be applied to the outside of the sclera without causing sloughing of any of the tissues.

The technic of insertion and subsequent removal of the seeds has been described and has been found to present no difficulties.

It is shown that a retinal glioma will disappear under the influence of radon seeds, and that in a concrete case a glioma of moderate size not only was not increased in size twenty-two months after the commencement of the treatment, but was shrunken and seemed to be completely scarred; it is, however, premature to say that the patient is cured.

Histologic changes in glioma cells that have been exposed to the radon seeds have been demonstrated.

Opacities of a special type in the lens have been seen to develop and afterward to disappear completely.

Further investigations with radon seeds on sarcomas as well as gliomas are being prosecuted, both intra-ocularly and as surface applications to the sclerotic.

The paper is a lengthy and important one, and should be read in the original.

W. ZENTMAYER.

Uvea

HETEROCHROMIA IRIDIS AND TUBERCULOUS DISEASES. V. ROSSI, Arch. di ottal. **38:** 367 (May-June); 371 (July-Aug.) 1931.

Admitting that the question of true heterochromia due to paralysis of the sympathetic nerve is not decided, Rossi considers the form with corneal precipitates and lens changes of inflammatory nature, in which tuberculosis may play an important part. He refers to Gradle's report in which, besides the cases of primary heterochromia, a number showing cyclitis and cataract were considered to be due to tuberculosis. Rossi reports four cases of tuberculous origin. In all of the cases the heterochromia was acquired and was accompanied by definite signs of chronic iridocyclitis, with posterior synechiae, occlusio pupillae or cataract. There were no changes suggesting sympathetic involvement. All of the cases showed signs of pulmonary or glandular tuberculosis, which in some cases was limited to roentgen changes at the apexes. The pigment changes in these cases are considered secondary to inflammation in the iris.

S. R. GIFFORD.

Therapeutics

ANTIGEN THERAPY IN OCULAR TUBERCULOSIS. G. GIVADE ARNOULD, Ann. d'ocul. **168:**624 (Aug.) 1931.

Mme. Arnould concludes as follows: Antigen therapy in ocular tuberculosis serves two purposes: (1) in diagnosis, and (2) in the treatment of tuberculosis.

In the diagnosis of external tuberculosis the Boquet-Negre reaction gives information of equal value to that of the Wassermann reaction in the diagnosis of syphilis. It is wise to repeat the reaction two or three times at intervals of from eight to ten days. With regard to treatment that was applied in a large number of cases, namely, one of chorioretinitis, thirteen of iritis, five of keratitis and one of interstitial keratitis, the results were excellent.

The local reaction in every case except one was satisfactory; the chorioretinitis disappeared; the crisis in the cases of iritis and keratitis was quieted; the cyclitic deposits were absorbed, and the disease conditions returned to normal. Unfortunately, we had only one observation pertaining to a tuberculous lesion of the inner eye. The general reaction was present in most of the cases; the patients increased in weight, their strength came back, and they seemed in better health, with normal temperatures. Cases in which the patients were obliged to stop work were exceptional.

S. H. MCKEE.

Society Transactions

EDITED BY DR. JOHN HERBERT WAITE

COLLEGE OF PHYSICIANS OF PHILADELPHIA, SECTION ON OPHTHALMOLOGY

Nov. 19, 1931

H. MAXWELL LANGDON, M.D., *Chairman*

A CASE OF IRITIS IN A FOUR MONTH OLD INFANT. DR. WILLIAM F. BONNER.

Except for a history of failure of the left pupil to react to light, the baby had always been healthy. The mother gave a history of septicemia following child birth. The blood count was normal, and the Wassermann reaction of the blood was negative. There was a plastic iritis on the left which did not respond to mydriatic and parenteral injections of protein.

A CASE OF BILATERAL OPTIC ATROPHY DUE TO METHYL ALCOHOL POISONING. DR. SHANNON.

Mr. H., aged 21, while visiting a garage, drank one-half glassful of what was supposed to be gin, on Thursday afternoon, May 1, 1931. Two hours later he suffered from acute gastritis with vomiting and severe pains in the abdomen. On the following morning he again vomited on four separate occasions, and on the same evening and through the night he vomited at frequent intervals. On Saturday morning, thirty-six hours after drinking the gin, he noticed for the first time that his vision was affected, and he was then rushed to the hospital; within a short time following his arrival, he noted a marked depreciation of vision. Shortly afterward he went completely blind. There has been no return of vision since. He visited my office on September 21, about four months later, and my examination revealed the following: There was no perception of light in either eye. The pupils were widely dilated, and were unresponsive to light. Rotation of the eyeballs was normal. Ophthalmoscopic examination showed the media to be clear; the disks were white, with deep glaucoma-like excavations. The arteries were narrowed. The maculae and periphery of the fundus exhibited no abnormalities of note. Tension, as measured by the Schiötz tonometer, was 15 mm. of mercury in the right eye and 18 mm. in the left.

It was impossible to secure any of the gin at the garage for an analysis, but by reason of the severe symptoms shortly following the ingestion of the gin by the patient, it seems reasonably safe to conclude that it must have contained methyl alcohol. Curiously, three of his companions imbibed as freely as he did. One suffered from abdominal distress, but in none of them was the vision affected. The interesting feature of the fundus picture was the extraordinary excavation of the disk.

The exact manner in which methyl alcohol acts on the internal organs is unknown, although once absorbed from the stomach or lungs into the blood, it is not so easily eliminated and burned up as ethyl alcohol. Apparently small and frequent doses tend to a cumulative effect in the system, resulting in serious damage to the digestive system, organs of sight and nervous system. According to Wood, there is a destructive inflammation of the optic nerve fibers or retinal elements (or both) followed by atrophy.

However, in this patient, the atrophy would suggest a primary form of a distinctly degenerative character.

DISCUSSION

DR. W. ZENTMAYER: Dr. Percy Friedenberg many years ago published his observations on the optic nerve head in methyl alcohol amaurosis, in which he described the condition present in the case shown by Dr. Shannon. In addition to the excavation, he found in one case an increase of glial tissue in the bottom of the cup, but in all the other instances there was an extrapapillary overgrowth of the connective tissue.

There is a difference of opinion as to the primary effect of the alcohol, whether it is on the ganglion cells of the retina or whether it is an effect on the nerve fibers or the interstitial substance. Jonas Friedenwald's experimental work indicates that the whole nerve is involved. A research Fellow of the Rockefeller Foundation has stated that if animals poisoned with methyl alcohol are kept in the dark, blindness does not supervene.

DR. G. E. DE SCHWEINITZ: I consider this a highly interesting paper. I have also noted in subjects with blindness caused by methyl alcohol excavation of the nerve head, simulating a glaucoma cup, originally described by Percy Friedenberg, and have reported one such case, but I have never observed the condition so perfectly developed as is evident in Dr. Shannon's patient.

The amount of wood alcohol capable of producing blindness varies considerably; from 2 to 5 drachms have caused blindness, as occurred in one of my own patients, while recovery after drinking one-half pint, or even larger quantities, has been observed. In short, intoxication caused by methyl alcohol is a typical example of idiosyncrasy.

The deadly effects of this poison may be due not only to the drinking of it, but also to inhalation and cutaneous absorption, as has been demonstrated experimentally. The blindness has been attributed to nutritional changes in the retinal ganglion cells (Ward Holden, Birch-Hirschfeld, Henry Smythe and others here and abroad), but the experimental results were not confirmed by other investigators (Jonas Friendenwald and A. Felty in Baltimore, Fewell and Perce de Long at the University of Pennsylvania). It is possible that the poison attacks the whole of the nerve.

The toxemia has also been attributed, not to the methanol itself, but to adulterants, for instance, fusel oil, and to imperfect combustion of the alcohol in the system and the production of formaldehyde. However, Reid Hunt's analyses have demonstrated that the chief toxicity belongs to the methyl alcohol. The whole subject from the experimental standpoint should be reviewed.

DR. T. B. HOLLOWAY: In my opinion, if these cases are taken as a group, idiosyncrasy is not the chief factor, but more important is the question of how much has been ingested and absorbed.

Generally you will find that those who have vomited freely, or when the stomach has been relieved of its contents soon after ingestion, do not suffer to the same extent as those who retain the poison.

While idiosyncrasy is doubtless a factor, the same is true in tobacco and ethyl alcohol poisoning, but doubtless not to the same extent. Dr. de Schweinitz has referred to the breaking down of the alcohol into other toxic substances, and it is my belief that when we definitely know the terminal products responsible for these symptoms, we shall be able to show whether the process begins in the ganglion cells, the nerve fibers or the interstitial tissue. I referred to the treatment in such cases in 1923, before the Section on Ophthalmology of the American Medical Association.

THE RECESSION OPERATION IN SQUINT. DR. LUTHER C. PETER.

Uncontrolled tenotomy has given way to the more rational recession suture, which is a safeguard against subsequent convergence insufficiency and the tendency to paralytic squint in the opposite direction. The operation of Jameson dates from 1922. Rae reported a recession suture at the International Congress of Ophthalmology in 1929. The new operation that I am about to report differs in many respects from the Jameson and the Rae operations. The several steps in the operation were devised without any knowledge of the Jameson method.

The important steps consist of the following: (1) a vertical incision of the conjunctiva 1 mm. back of the point of insertion; (2) subconjunctival dissection toward the canthus; (3) introduction of a tendon hook with slight freeing of the muscle insertion from the capsule; (4) a double-armed whip stitch introduced near the upper and lower borders close to the muscle hook, from without in, so as to grasp the muscle properly and spread it on the sclera; (5) cutting of the tendon, leaving 1 mm. of stump; (6) 2 mm. bites in the superficial sclera, widely separated, so as to spread the tendon on the sclera, and precisely placed so as to avoid torsion; (7) passing of the suture through the center of the tendon stump, 3 mm. apart, and through the conjunctiva anterior to the stump; (8) tying of the suture with a loop, after drawing up the tendon to its proper place; (9) closing of the conjunctiva and sclera by a running, untied suture. The conjunctival suture is removed on the fifth day, and the scleral suture on the seventh.

The essential difference between this and the Jameson suture lies in the use of no. 1 silk, instead of catgut, which admits a very fine needle and consequently offers less trauma to the sclera. All the strain of the suture is placed on the tendonous stump. Closure of the suture by means of a loop admits of modification on the second or third day if necessary.

The indications for the operation, as compared with the shortening operations on the weak muscle, are briefly as follows: (*a*) In the correction of moderate degrees of squint, advancement of the long muscle should be given the preference on the basis of sound underlying surgical principles. In squint of high degrees, or in such instances in which more than one surgical procedure becomes necessary, the recession

suture logically becomes a supplementary operation of much value. (*b*) In esophoria and exophoria, the choice of operations is largely determined by the ratio of abduction to adduction. When abduction is excessively low, it is wise to increase the power of the external rectus. Conversely, if adduction is excessive and abduction is normal, a recession of the internal rectus is indicated.

DISCUSSION

DR. W. S. REESE: I think that this paper is timely, and I should like to induce some of the members of the section to do this operation. I have been doing it for the past ten years, and it has worked out very well. I think that it is an advantage to select a technic that requires only one scleral stitch, as there have been several unfortunate cases reported of perforation of the globe. There is slight danger of this if one is careful to place the stitch so superficially that the needle can be seen passing through the sclera. This is facilitated if moderate pressure is made on the globe.

DR. L. C. PETER: Dr. Peter thanked Dr. Reese for his courtesy, and added that he was perfectly right that one need have no fear of perforation of the sclera by the needle. The needle should be a fully curved, very small one, and the point should be visible at all times; in this way there is no danger of perforation of the sclera.

OPHTHALMOLOGIC IMPORTANCE OF FOCAL INFECTIVE PROSTATITIS. DR. P. S. PELOUZE (by invitation).

This article appeared in full in the March, 1932, issue of the ARCHIVES, page 372.

DISCUSSION

DR. G. E. DE SCHWEINITZ: I should like to refer to some of my experiences with nongonorrheal prostatitis as an etiologic factor in ocular disorders, iritis and certain forms of relapsing keratitis, published some years ago, to Dr. Swinburne's contention that these forms of prostatitis should be regarded equally with chronic infection of the tonsil and to Dr. Zentmayer's excellent paper on this subject.

I am glad that Dr. Pelouze has brought the results of his large experience to the attention of the Section. I differ somewhat with his opinion as to gonococcal iritis, which is a perfectly well established form of iridocyclitis, chiefly plastic in type, which may be associated with or follow arthritis, owing to the influence of gonococci (or perhaps their toxins) on the anterior uveal tract; indeed, in these circumstances the gonococci have been recovered from the anterior chamber by Sidler-Huguenin. This form of iridocyclitis is favorably influenced by the administration of large doses of gonococcus vaccine, as has been demonstrated in clinical reports published here and abroad.

DR. W. ZENTMAYER: In 1926 I presented a paper entitled "The Prostate as a Remote Focus of Infection in Ocular Inflammations" before the Section on Ophthalmology of the American Medical Association. My object in so doing was to direct attention anew to the importance of searching further than the mouth, nose and throat for the portal of infection in cases of metastatic infection, and to emphasize that it is not infrequently to be found in the prostate gland.

At the time this paper was presented, the only other two papers on the subject in the ophthalmic literature were one by Thomasson and one by Dernehl.

In that paper the points brought out by Dr. Pelouze, of the danger of manipulation of the prostate during the time metastatic inflammation is active and the fact that the prostatic infection is more often non-gonococcal than gonococcal, were emphasized.

Dec. 17, 1931

H. MAXWELL LANGDON, M.D., *Chairman*

REMOVAL OF OXIDIZED TISSUE REMAINING AFTER FOREIGN BODY OF THE CORNEA. DR. GEORGE H. CROSS.

Dr. Cross read a paper in which he said that corneal burrs are used to remove the brownish-stained tissue that remains in the cornea following the removal of hot metallic foreign bodies. They are also used to remove the undercut edge of the crater of a foreign body which is so annoying to the patient that it often causes him to return in a short time, complaining that all the foreign body was not removed. This edge provides an excellent harbor for bacteria that may produce ulcers. The burrs are useful to smooth out the roughened surface area from which a foreign body has been removed. Originally burrs were soldered in a metal handle and thus were too expensive to throw away.

LUPUS ERYTHEMATOSUS OF THE CONJUNCTIVA AND EYELIDS. DR. JOSEPH V. KLAUDER and DR. PERCE DE LONG.

This article will appear in full in a later issue of the ARCHIVES.

LYMPHOMAS OF THE CONJUNCTIVA. DR. C. E. G. SHANNON and (by invitation) DR. L. F. MCANDREWS.

The presenters showed a case of bilateral lymphomas of the conjunctiva. The patient, a woman, aged 56, noticed small masses appear beneath the bulbar conjunctiva, near the insertion of the superior rectus muscle of each eye. These masses had been present for about one year. The mass in the left eye gradually increased in size, until it involved the upper half of the globe. In the right eye, the masses remained stationary. General physical examination and examination of the blood gave normal results. The diagnosis was made by microscopic examination. Radium was used to remove the growths. Four applications were given with excellent results. This is the first case to be reported in which radium was used to remove conjunctival lymphomas. A complete review of this subject was also presented. At the present time, there is no satisfactory explanation as to the etiology of lymphomas of the orbit.

DISCUSSION

DR. B. L. CRAWFORD: Lymphomas of the conjunctiva have a characteristic histologic picture. The lymphomas in this location seem to be more definitely neoplastic than those in other

locations of the body. The diagnosis of lymphoma is frequently made of lesions in other parts of the body where there is a more or less indefinite collection of lymphoid type of cells without true tumor formation. This neoplasm, as illustrated by the lantern slide, is composed of a fairly uniform type of cell which closely resembles the lymphoid cell, rather loosely arranged with little stroma and numerous small blood vessels. In differentiating the lymphomas from round cell sarcomas, there are several points to be considered, as follows: malignant tumors are rarely multiple as in this instance; the uniformity of the type of cell, the lack of irregularity of the cells, the absence of mitosis of the nuclei usually seen in malignant tumors, the type of blood vessels in the stroma and the presence of a fairly definite capsule are important. Leukemic infiltration is excluded by a careful examination of the blood.

DR. WILLIAM ZENTMAYER: Are not the blood vessels in lymphomas different from those in a malignant tumor?

DR. B. L. CRAWFORD: The small blood vessels in the stroma of the lymphoma had well defined walls and were not in such an intimate relation with the tumor cells as in a sarcoma.

DR. WILLIAM ZENTMAYER: Through the courtesy of Dr. Shannon I saw this patient before treatment was started. With the history and negative results of a physical examination and an examination of the blood, I suggested the possibility of its being a case of amyloid degeneration of the conjunctiva. I have no apology to offer, as in the pathologic report in one of the cases hyaline degeneration was noted as a feature.

Book Reviews

Juvenile Amaurotic Idiocy. Research Concerning the Clinical and Hereditary Aspects. By Torsten Sjögren from the State Institute for Racial Biology, Uppsala. Price, 15 S. crowns. Pp. 425. Lund: R. Larsson, 1931.

The author has given in German a careful and detailed study from the clinical and the hereditary point of view of that form of juvenile idiocy which Vogt in 1906 differentiated from the better known infantile form of Tay-Sachs' disease. The typical clinical picture was that of one or more previously healthy children in one family, who became affected, generally during the school years, from the ages of 6 to 14. The first symptom is usually failing vision, which leads to complete blindness in the course of months. Several children of the same family may become affected in the same age year. The disease starts gradually. Ophthalmoscopic examination shows atrophic disks. Mental development is arrested and then retrogresses. The disease progresses little by little to deepest psychic dementia, marasmus and death in the course of from five to ten years. The motor functions also diminish steadily, leading finally to complete paralysis, which may be atonic or spastic. There are no data suggesting a syphilitic inheritance. With this form Vogt associated the familial cerebral diplegias that were described by Pelizaeus (1885), Freud (1893) and Higier (1896). As to the first form, the clinical symptoms differ markedly from those generally considered as typical of juvenile amaurotic idiocy, and Merzbacher (1910) showed that there was a special, quite characteristic underlying histopathology. Spielmeyer (1906) was the first to report the histologic, pathognomonic basis, i. e., Schaffer's cellular changes. According to Schob and Sjögren, the disease generally begins with the second dentition, i. e., at the age of from 4 to 6. It is unusual after the tenth year. Very rarely there is a previous history of mental backwardness. The first symptom is usually a gradual but fairly rapid loss of vision, leading to blindness and lessening of intelligence. Epileptic attacks are observed at this stage, or may not come until some years later. In contradistinction to the infantile form, the ophthalmoscope shows a rather variable picture, generally that of retinitis pigmentosa, or of chorioretinitis with secondary optic atrophy.

One notes "slight atrophy, with a washed out, indistinct nerve head. All the vessels, but especially the arteries, are markedly contracted. In both eyes there are macular changes, and toward the periphery, beginning rather near the center of the fundus, there is a diffuse brownish stippling on a reddish-gray background, with contrasting, washed out, paler areas" (Rogalski, 1910). Or, "The disk is snow-white and sharply defined, and the arteries are markedly, and the veins somewhat, contracted. A dirty red spot is present at the macula. The peripheral fundus is albinotic. Small, light yellow areas alternate with black pigment granules, which are sharply defined, and which seem to be sown

broadcast over the fundus. There is no sign of a bone corpuscle arrangement of the pigment" (Kuffler, 1912). Again, "There is bilateral atrophy of the retina and nerve. There is an indistinct disk margin. The vessels, especially the arteries, are greatly contracted. There is a diffuse pigment, stippled deposit (left eye), with a few larger granules in the periphery (right eye)" (Schob, 1912). There are diffuse peripheral pepper and salt deposits. Occasionally, the pigment stippling is marked in the central, foveal part of the fundus, the vessels are not markedly contracted or the disk is of a muddy, gray color. Or, "Small areas of old choroiditis are found scattered among, but not encircled by, pigment granules." There is a pale, atrophic disk with narrowed vessels. There is a white patch at the macula, surrounded by a red zone. The peripheral retina is atrophic and is covered with fine pigment granules. In some cases there was optic atrophy alone, and in a very few no ophthalmoscopic changes in the fundus in spite of the marked amaurosis, which may have been due to pathologic changes in the central or peripheral neurons, as suggested by Spielmeyer. Walter (1918) reported a remarkable atypical case with a normal fundus and good vision.

The mental disturbance is an extraordinarily constant symptom. The children, who generally have developed normally until they reached school age, become dull, listless and incapable of assimilating teaching; in a word, they show a progressive, psychic deterioration. They are not infrequently irritable, or lacrimose, as well. Later, they lose what little knowledge they have acquired, and thus, the ability to read (braille type). Speech too is affected and, finally, is completely lost. They cannot dress or undress themselves or attend to the simple needs of nature; they fail to recognize persons in their immediate surroundings; they become filthy, and they finally show a picture of absolute idiocy. Hallucinations and delusions are generally absent. Epileptic attacks, which are of no special interest as to type and course, are observed later. Schob has called attention to a certain ataxia and lack of security in gait, which may progress and become the dominant symptom of the disease. There is good motility in the sitting or lying position, with short, tripping steps in walking. Vogt noted unsteady gait, with a marked forward inclination of the body, and stiff legs, which move with difficulty. Finally, all locomotion is impossible, even with support. The patients lie helpless in bed. In the terminal stages, a spastic paralysis is generally seen. The tendon reflexes are usually brisk or accentuated. Syphilis seems to play no rôle whatever. The serologic findings are normal. Death generally supervenes between the ages of 14 and 16, often with marked wasting, although a few patients live to be from 23 to 25 years of age.

In the infantile form, there is a marked familial occurrence, but this occurs predominantly in stocks of Aryan racial origin. A transitional late infantile form has been described without the typical macular changes, and with a less uniform picture of psychopathology. As the changes in the fundus are variable and the psychic degeneration is not characteristic, the diagnosis is often hypothetic. The familial occurrence and data of consanguinity have suggested a recessive inheritance. Kufs is of the opinion that various diseases of the fundus, retinitis pig-

mentosa and its variants, progressive familial macular heredodegeneration, certain forms of hereditary optic atrophy, recessive deaf mutism and "nervous deafness" represent, with the various forms of amaurotic idiocy, a nosological unity. The hereditary disease that is basic to all the organic lesions described has the tendency to appear in various phenotypes.

Sjögren draws the following conclusions: Juvenile amaurotic idiocy is not so rare in Sweden as it is in other countries. Ophthalmoscopic signs and the development and form of neurologic symptoms are uniform and constant in their progressive course. The extrapyramidal symptoms, motor disturbances, abnormal gait (marche à petits pas, démarche trépidante), increasing tendency to squatting and hypertonia of a predominantly rigid type are stressed. A recessive monohybrid inheritance is probable, in contradistinction to the inheritance of the infantile form. The ancestors who were probably heterozygotic show a definite tendency to multiplicity in several circumscribed localities. Statistics as to complication with dementia praecox, oligophrenia and epilepsy among the brethren of ancestors show a high probability quota.

PERCY FRIDENBERG.

Fifth Annual Report of the Giza Ophthalmic Laboratory, Cairo, Egypt, 1930. Dr. Rowland P. Wilson, Director. Pp. 102. Cairo, Egypt, Schindler Press, 1931.

This report by the director of the Government Ophthalmic Laboratory at Cairo, Egypt, describes the clinical, educational and experimental work during the year. Besides other pathologic specimens, 118 globes were examined histologically. The reports are illustrated by excellent photomicrographs, and include tuberculosis of the lacrimal gland, gumma of the lid, nevus of the caruncle, tuberculosis of the conjunctiva, angioma of the conjunctiva and fly infestation of the orbit, as some of the rarer conditions. Seven cases of amyloidosis of the conjunctiva following trachoma were examined. Over 1,000 bacteriologic examinations were made.

Postgraduate courses lasting a month for medical officers of the public health department are held twice a year; they are well attended, as both courses are required of candidates for the service.

A number of clinical cases were referred to the laboratory from the Ophthalmic Hospital at Giza and other hospitals for examination with the slit-lamp, fundus photography and other special investigation.

Research was conducted chiefly on the subject of trachoma. A survey on trachoma previously reported by the typical village of Bahtim was continued, especially to determine the age at which infants became infected. Of thirty-seven born between September and December, definite trachoma developed in all but three between the ages of 10 weeks and 11 months, the average age being from 7 to 8 months. Of nineteen born between April and August, the disease developed in all but three within seven months, the average age being 4½ months. Thus, trachoma developed in the children born in the hot months several months earlier than in those born in the winter. In 65 per cent of the cases the onset of trachoma was preceded by an acute Koch-Weeks conjunctivitis.

In the treatment of trachoma, a combination of 10 per cent copper sulphate and chaulmoogra oil was tried in seventy cases, and the results seemed more rapid and satisfactory than were obtained with either drug alone.

In vernal conjunctivitis, radium therapy was found superior to other methods, including treatment with afenil and lactic acid. From 300 to 600 milligram hours was given over from two and one-half to five months.

A detailed report is given of the work on the etiology of trachoma, some of which was performed with the assistance of Dr. Thygeson of Denver. No cultures of Bacillus granulosis were obtained. Some stains of a bacillus morphologically similar to B. granulosis were found, but all failed to agglutinate with specific serum. Thirteen monkeys inoculated with B. granulosis from various sources, some of which were performed by Dr. Thygeson, were negative. With fresh tissue from Dr. Thygeson's infected monkeys, however, a picture was produced which resembled that seen in his monkeys. Four human inoculations with B. granulosis were negative. It was concluded that the disease produced in monkeys has not been proved to be trachoma, and that a judgment of the etiologic importance of B. granulosis must await further human inoculations.

S. R. GIFFORD.

An Introduction to the Literature of Vertebrate Zoology. By Casey A. Wood, M.D., LL.D., Collaborator, Division of Birds, Smithsonian Institution. Price, $15. Pp. 643, with a colored frontispiece. London: Oxford University Press, 1931.

This book is based on a careful survey of the McGill University Libraries. The first part of the work is devoted to a readable and excellent digest of the significant literature from Aristotle to the present. This digest is interesting and valuable because the author has viewed the various works historically and geographically as well as to subject and source, and also because many fundamental treatises in vertebrate zoology are brought to the reader's attention. The second section is a student's and librarian's index to the final division of the treatise, which consists of a partially annotated catalog of authors and titles of printed books, manuscripts, periodicals, etc., pertaining to vertebrate zoology.

While using this work, one must always keep in mind that it represents only an introduction to the vast literature on vertebrate zoology. The treatise, nevertheless, is an important aid in filling an urgent demand in the realm of vertebrate literature. It is worthy of much consideration by all students and research workers interested in historical zoology, and will doubtless be welcomed by all who desire an acquaintance with zoological literature. It should find a ready place in medical as well as in zoological libraries, not only because it surveys much vertebrate anatomic literature, but because it portrays the intimate relationship of medicine in fostering zoological research, in that many of the investigators in this field of zoology have been physicians. The compiler is to be commended for his scholarly production and courage in initiating and completing such an endeavor.

OLIN E. NELSEN.

Ocular Therapeutics. By Sanford R. Gifford, M.D. Price, $3.25 net. Pp. 272. Philadelphia, Lea & Febiger, 1932.

Dr. Gifford first considers such matters as equipment, anesthetics and their administration, the drugs used in ophthalmology and specific and nonspecific protein therapy. An entire chapter is devoted to the important subject of physical therapy. The rest of the book takes up in turn the treatment of the various well defined ocular diseases and disturbances.

The result is an astonishing amount of information, presented concisely and yet in sufficient detail so that further recourse to original articles will be but rarely necessary. Recent measures receive their full share of attention, and, though there are undoubted omissions, no serious fault can be found in the matter of completeness. However, it is not as a mere compendium of information that the book is of most value. Perhaps the chief virtue lies in the author's thoroughly sane and rational appraisal of the various therapeutic means and procedures considered. The book is pervaded throughout by good sense and so cannot fail to be useful.

O. P. P.

Ophthalmo-und Oto-Neurologie. By Dr. E. A. Spiegel and Dr. Ignaz Sommer. Price, 29.60 marks. Pp. 366. Berlin: Julius Springer, 1931.

The first 200 pages of this excellent book are concerned with neurology of the eye. The anatomy of the brain and cord is described in the first chapter, and the various fiber tracts are clearly depicted by original diagrams. The second chapter considers the cerebrospinal fluid, its origin and circulation, and the diagnostic significance of lumbar puncture in diseases of the eye and the ear. Chapter three describes the optic pathways and centers. The subject is brought up to date by including the work of Brower, Pfeiffer and Henchen. A short résumé of the physiology and pathology of the visual fields is then given. Chapter four summarizes the principal ophthalmoscopic appearances of the fundus in disease of the nervous system and of the ear. This chapter will probably prove of more value to otologists and neurologists than to the ophthalmologist. Chapter five deals with the external ocular muscles and their paralyses. The sections on the cortical centers for ocular movements, associate paralyses and optokinetic nystagmus are extremely well done. Chapter six describes the pupil reflexes and their disturbances. A short account of the sensory nerve supply of the eye and its disturbances closes the part of the book that deals primarily with the eye.

In the last half of the book, the section on nystagmus will be of interest to ophthalmologists, as some of our ideas of the slow and quick components of nystagmus are based on the experimental researches of Spiegel.

The book is a welcome addition to the textbooks of ophthalmology, and it is hoped that an English translation will soon appear to make the book available to those not conversant with German.

F. H. ADLER.

Directory of Ophthalmologic Societies *

INTERNATIONAL

International Ophthalmologic Congress

Secretary: Dr. E. Marx, Rotterdam, Holland.
Place: Madrid, Spain. Time: 1933.

FOREIGN

British Medical Association, Section on Ophthalmology

President: Sir J. Herbert Parsons, 54 Queen Anne St., Cavendish Square, London, W. I.
Place: London. Time: July 27-29, 1932.

Ophthalmological Society of the United Kingdom

President: Mr. A. H. H. Sinclair, 6 Charlotte Square, Edinburgh.
Secretary: Mr. Humphrey Neame, 56 Portland Place, London, W. I.
Place: Edinburgh. Time: May 12-14, 1932.

Oxford Ophthalmological Congress

Master: Dr. Cyril Walker, Bristol, England.
Secretary: Dr. C. G. Russ-Wood, 12 St. John's Hill, Shrewsbury, England.
Place: Oxford. Time: July 7-9, 1932.

Royal Society of Medicine, Section of Ophthalmology

President: Dr. E. W. Brewerton, 73 Harley St., W. 1, London.
Secretary: Dr. P. G. Doyne, 60 Queen Anne St., W. 1, London.
Place: London. Time: June 10, 1932.

Société Française d'Ophthalmologie

Secretary: Dr. René Onfray, 6 avenue de la Motte Picquet, Paris 7è.
Place: Paris. Time: July 17-21, 1932.

German Ophthalmological Society

Secretary: Prof. A. Wagenmann, Heidelberg.
Place: Leipzig. Time: May 19-21, 1932.

NATIONAL

American Medical Association, Section on Ophthalmology

Chairman: Dr. Harry Friedenwald, 1212 Eutaw Pl., Baltimore.
Secretary: Dr. Parker Heath, 1551 Woodward Ave., Detroit.
Place: New Orleans. Time: May 11-13, 1932.

American Academy of Ophthalmology and Oto-Laryngology

President: Dr. S. Hanford McKee, 1528 Crescent St., Montreal, Canada.
President-Elect: Dr. B. R. Shurley, 62 W. Adams Ave., Detroit.
Executive Secretary: Dr. William P. Wherry, Medical Arts Bldg., Omaha.
Place: Montreal, Canada. Time: September 19-23, 1932.

American Ophthalmological Society

President: Dr. Edward C. Ellett, 130 Madison Ave., Memphis, Tenn.
Secretary: Dr. Emory Hill, Professional Bldg., Richmond, Va.
Place: New London, Conn. Time: June 27-29, 1932.

* Secretaries of societies are requested to furnish the information necessary to make this list complete and to keep it up to date.

SECTIONAL

New England Ophthalmological Society

President: Dr. David W. Wells, Hotel Westminster, Boston.
Secretary-Treasurer: Dr. Theodore L. Terry, 243 Charles St., Boston.
Place: Massachusetts Eye and Ear Infirmary, 243 Charles St., Boston. Time: 8 p. m., third Tuesday of each month from November to April, inclusive.

Pacific Coast Oto-Ophthalmological Society

President: Dr. W. F. Hoffman, 317 Summit Ave., Seattle.
Secretary-Treasurer: Dr. Frank Friesen, 1208 Roosevelt Bldg., Los Angeles.
Place: Seattle. Time: June 30-July 2, 1932.

Puget Sound Academy of Ophthalmology and Oto-Laryngology

President: Dr. John H. Harter, 1215 Fourth Ave., Seattle.
Secretary-Treasurer: Dr. Francis A. Brugman, 806 Cobb Bldg., Seattle.
Time: 8 p. m., third Tuesday of each month, except July and August.

Sioux Valley Eye and Ear Academy

President: Dr. John B. Gregg, 101 S. Main St., Sioux Falls, S. Dakota.
Secretary-Treasurer: Dr. F. H. Roost, 420 Trimble Bldg., Sioux City, Iowa.
Place: Sioux City, Iowa. Time: November, 1932.

Southern Medical Association, Section on Eye, Ear, Nose and Throat

Chairman: Dr. Wiley R. Buffington, 211 Camp St., New Orleans.
Secretary: Dr. William D. Gill, 323 Medical Arts Bldg., San Antonio, Texas.
Place: Birmingham, Ala. Time: November, 1932.

Southwestern Michigan Triological Society

President: Dr. W. E. McGarvey, 802 Jackson City Bank Bldg., Jackson.
Secretary: Dr. Don C. Lyons, 1405 Union and Peoples National Bank Bldg., Jackson.
Time: Third Thursday of alternate months.

STATE

Colorado Ophthalmological Society

President: A presiding officer is selected for each meeting alternately until all members have served.
Secretary: Dr. Ralph W. Danielson, 258 Metropolitan Bldg., Denver.
Place: Capitol Life Building, Denver. Time: 7:30 p. m., third Saturday of the month, October to April, inclusive.

Connecticut State Medical Society, Section on Eye, Ear, Nose and Throat

President: Dr. M. H. Merriman, 115 Prospect St., Waterbury.
Secretary-Treasurer: Dr. Walter L. Hogan, 750 Main St., Hartford.
Time: May, November.

Eye, Ear, Nose and Throat Club of Georgia

President: Dr. W. L. McDougall, 50 Whitehall St., S. W., Atlanta.
Secretary-Treasurer: Dr. W. O. Martin, Jr., 412 Medical Arts Bldg., Atlanta.

Indiana Academy of Ophthalmology and Oto-Laryngology

President Dr. J. R. Gillum, 221 S. Sixth St., Terre Haute.
Secretary: Dr. J. Kent Leasure, 23 E. Ohio St., Indianapolis.

Iowa Academy of Ophthalmology and Oto-Laryngology

President: Dr. Royal French, 28 S. First Ave., Marshalltown.
Secretary-Treasurer: Dr. W. F. Boiler, 105 E. Iowa Ave., Iowa City.

MICHIGAN STATE MEDICAL SOCIETY, SECTION OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Dr. Wilfred Haughey, 303 Post Bldg., Battle Creek.
Secretary: Dr. H. O. Westervelt, 239 Pipestone St., Benton Harbor.

MINNESOTA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Douglas F. Wood, 74 S. Ninth St., Minneapolis.
Secretary-Treasurer: Dr. Walter E. Camp, Medical Arts Bldg., Minneapolis.
Time: Second Friday of each month from October to May.

MONTANA ACADEMY OF OTO-OPHTHALMOLOGY

President: Dr. L. H. Huber, Livingston.
Secretary: Dr. A. W. Morse, Phoenix Bldg., Butte.

NEW JERSEY STATE MEDICAL SOCIETY, SECTION ON OPHTHALMOLOGY, OTOLOGY AND RHINOLARYNGOLOGY

Chairman: Dr. H. L. Harley, 101 S. Indiana Ave., Atlantic City.
Secretary: Dr. B. E. Failing, 31 Lincoln Park, Newark.
Place: Atlantic City. Time: June 15-17, 1932.

NEW YORK STATE MEDICAL SOCIETY, EYE, EAR, NOSE AND THROAT SECTION

Chairman: Dr. R. T. Atkins, 4 W. Fifty-Third St., New York.
Secretary: Dr. D. F. Gillette, 109 S. Warren St., Syracuse.
Place: Buffalo. Time: May 23-24, 1932.

NORTH DAKOTA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. F. L. Wicks, Valley City.
Secretary-Treasurer: Dr. W. L. Diven, National Bank Bldg., Bismarck.

OREGON ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. H. M. Hendershott, 193 Eleventh St., Portland.
Secretary-Treasurer: Dr. Andrew J. Browning, 418 Mayer Bldg., Portland.
Place: Good Samaritan Hospital. Time: Third Tuesday of each month.

RHODE ISLAND OPHTHALMOLOGICAL AND OTOLOGICAL SOCIETY

President: Dr. Frank W. Dimmitt, 195 Thayer St., Providence.
Secretary: Dr. N. A. Bolotow, 108 Waterman St., Providence.
Place: Rhode Island Medical Library. Time: 8:30 p. m., second Thursday in October, December, February and April.

SOUTH CAROLINA MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. W. B. McWhorter, 106 E. Earle St., Anderson.
Secretary-Treasurer: Dr. G. M. Truluck, Orangeburg.
Time: April and December.

TEXAS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President: Dr. W. D. Jones, 3116 Live Oak St., Dallas, Texas.
Secretary: Dr. A. F. Clark, 1034 Nix Professional Bldg., San Antonio.

UTAH OPHTHALMOLOGICAL SOCIETY

President: Dr. D. W. Henderson, 75 S. Main St., Salt Lake City.
Secretary-Treasurer: Dr. T. F. Welch, 14 Exchange Pl., Salt Lake City.

VIRGINIA SOCIETY OF OTO-LARYNGOLOGY AND OPHTHALMOLOGY

President: Dr. C. P. Jones, Newport News.
Secretary-Treasurer: Dr. H. G. Preston, 307 Professional Bldg., Harrisonburg.

WEST VIRGINIA STATE MEDICAL ASSOCIATION, EYE, EAR, NOSE AND THROAT SECTION

President: Dr. G. A. Smith, Montgomery.
Secretary: Dr. O. H. Bobbitt, Medical Arts Bldg., Charleston.

LOCAL

Academy of Medicine of Northern New Jersey, Section on Eye, Ear, Nose and Throat

Chairman: Dr. Charles W. Buvinger, 50 Washington St., East Orange.
Secretary: Dr. Earl LeRoy Wood, 192 Roseville Ave., Newark.
Place: 91 Lincoln Park South. Time: 8:45 p. m., second Monday of each month.

Academy of Medicine of Toronto, Section of Ophthalmology

Chairman: Dr. P. J. F. Houston, 34 Hurndale Ave., Toronto.
Secretary: Dr. Alexander E. MacDonald, 151 Bloor St., W., Toronto.
Time: First Monday of winter months.

Atlanta Eye, Ear, Nose and Throat Society

President: Dr. L. C. Rouglin, 127 Peachtree St., N. E., Atlanta, Ga.
Secretary: Dr. William O. Martin, Jr., Medical Arts Bldg., Atlanta, Ga.
Place: Academy of Medicine, 38 Prescott St., N. E. Time: 8 p. m., last Thursday of each month.

Baltimore Medical Society, Section on Ophthalmology

Chairman: Dr. Harry Friedenwald, 1212 Eutaw Pl., Baltimore.
Secretary: Dr. William M. Rowland, 906 Cathedral St., Baltimore.
Place: Medical and Chirurgical Faculty, 1211 Cathedral St. Time: 8:30 p. m., fourth Thursday of each month from October to May.

Brooklyn Ophthalmological Society

President: Dr. John N. Evans, 23 Schermerhorn St., Brooklyn.
Secretary: Dr. Michael J. Buonaguro, 589 Lorimer St., Brooklyn.
Place: Kings County Medical Society Bldg., 1313 Bedford Ave. Time: Third Thursday in February, April, May, October and December.

Buffalo Ophthalmologic Club

President: Dr. Joseph C. O'Gorman, 1324 Jefferson Ave., Buffalo.
Secretary-Treasurer: Dr. Thurber Le-Win, 112 Linwood Ave., Buffalo.

Chicago Ophthalmological Society

President: Dr. Frank Brawley, 30 N. Michigan Ave., Chicago.
Secretary: Dr. Richard C. Gamble, 30 N. Michigan Ave., Chicago.
Place: Medical and Dental Arts Club. Time: Third Monday of each month from October to May.

Cleveland Academy of Medicine, Section of Ophthalmology and Oto-Laryngology

Chairman: Dr. M. W. Jacoby, 2323 Prospect Ave., Cleveland.
Secretary: Dr. H. C. Rosenberger, 1065 Rose Bldg., Cleveland.
Place: Winton Hotel. Time: Fourth Friday of each month.

Cleveland Ophthalmological Club

Chairman: Dr. Roy B. Metz, Hanna Bldg., Cleveland.
Secretary: Dr. Paul G. Moore, Medical Arts Bldg., Cleveland.

College of Physicians, Philadelphia, Section on Ophthalmology

Chairman: Dr. L. F. Appleman, 308 S. Sixteenth St., Philadelphia.
Clerk: Dr. Alexander G. Fewell, 1924 Pine St., Philadelphia.
Time: Third Thursday of every month from October to April, inclusive.

Columbus Ophthalmological and Oto-Laryngological Society

Chairman: Dr. Ivor Clark, 188 E. State St., Columbus, Ohio.
Secretary-Treasurer: Dr. Francis W. Thomas, 327 E. State St., Columbus, Ohio.
Time: First Monday of each month.

Dallas Academy of Ophthalmology and Oto-Laryngology

President: Dr. L. M. Sellers, 717 Pacific Ave., Dallas, Texas.
Executive Secretary: Dr. W. Mood Knowles, 717 Pacific Ave., Dallas, Texas.
Place: Dallas Athletic Club. Time: 6:30 p. m., first Tuesday of each month from October to June. The November, January and March meetings are devoted to clinical work.

Des Moines Academy of Ophthalmology and Otolaryngology

President: Dr. Cecil C. Jones, 1121 Equitable Bldg., Des Moines, Iowa.
Secretary-Treasurer: Dr. Grace Doane, 614 Bankers Trust Bldg., Des Moines, Iowa.
Time: 7:45 p. m., third Monday of every month, September to May.

Detroit Ophthalmological Club

Chairman: Members rotate alphabetically.
Secretary: Dr. Raymond S. Goux, David Whitney Bldg., Detroit.
Time: 6:30 p. m., first Wednesday of each month.

Eastern New York Eye, Ear, Nose and Throat Association

President: Dr. F. M. Sulzman, 1831 Fifth Ave., Troy.
Secretary-Treasurer: Dr. Arthur F. Holding, 142 Washington Ave., Albany.
Time: Third Wednesday in October, November, March, April, May and June.

Fort Worth Eye, Ear, Nose and Throat Society

President: Dr. R. H. Needham, 1304 Medical Arts Bldg., Fort Worth, Texas.
Secretary-Treasurer: Dr. Van D. Rathgeber, 1011 Medical Arts Bldg., Fort Worth.
Place: University Club. Time: 6:30 p. m., first Friday of each month except July and August.

Grand Rapids Eye, Ear, Nose and Throat Society

President: Dr. Carl F. Snapp, 26 Sheldon Ave., Grand Rapids, Mich.
Secretary-Treasurer: Dr. Walter W. Oliver, 26 Sheldon Ave., Grand Rapids, Mich.
Place: Various local hospitals. Time: Third Thursday of alternating months, September to May.

Houston Academy of Medicine, Eye, Ear, Nose and Throat Section

President: Dr. L. W. Raney, 609 Fannin St., Houston, Texas.
Secretary: Dr. J. F. Gamble, 201 Main St., Houston, Texas.
Place: Medical Arts Bldg., Harris County Medical Society Rooms. Time: 8 p. m., first Thursday of each month from September to June.

Indianapolis Ophthalmological and Oto-Laryngological Society

President: Dr. B. J. Larkin, 23 E. Ohio St., Indianapolis.
Secretary: Dr. Kenneth L. Craft, Hume-Mansur Bldg., Indianapolis.
Place: University Club. Time: 6:30 p. m., second Thursday of each month from October to May.

Kansas City Society of Ophthalmology and Oto-Laryngology

President: Dr. W. E. Keith, 1103 Grand Ave., Kansas City, Mo.
Secretary: Dr. O. S. Gilliland, Professional Bldg., Kansas City, Mo.
Time: Third Thursday of each month from September to May. The November, January and March meetings are devoted to clinical work.

Long Beach Eye, Ear, Nose and Throat Society

President: Dr. R. R. Montgomery, 102 Pine Ave., Long Beach, Calif.
Secretary-Treasurer: Dr. Russell T. Uhls, 823 Security Bldg., Long Beach, Calif.
Place: Pacific Coast Club. Time: Last Wednesday of each month from September to June.

Los Angeles County Medical Society, Section on Ophthalmology and Oto-Laryngology

President: Dr. J. Frank Friesen, 1208 Roosevelt Bldg., Los Angeles.
Secretary-Treasurer: Dr. F. H. Brandt, 1052 W. Sixth St., Los Angeles.
Place: Mary Louise Cafe. Time: 6:30 p. m., fourth Monday of each month from October to May, inclusive.

Medical Society of the District of Columbia, Section of Ophthalmology and Oto-Laryngology

Chairman: Dr. James A. Flynn, 1511 Rhode Island Ave., Washington.
Secretary: Dr. E. R. Gookin, Medical Science Bldg., Washington.
Place: 1718 M St., N. W. Time: 8 p. m., third Friday of each month.

Memphis Society of Ophthalmology and Oto-Laryngology

Chairman: Each member, in alphabetical order.
Secretary: Dr. R. O. Rychener, Exchange Bldg., Memphis, Tenn.
Place: Eye Clinic of Memphis Eye, Ear, Nose and Throat Hospital. Time: 8 p. m., second Tuesday of each month. Annual meeting, Jan. 10, 1933.

Milwaukee Oto-Ophthalmic Society

President: Dr. T. F. McCormick, 324 Wisconsin Ave., Milwaukee.
Secretary-Treasurer: Dr. O. P. Schoofs, 324 E. Wisconsin Ave., Milwaukee.
Place: University Club. Time: 6:30 p. m., third Tuesday of each month.

Nashville Academy of Ophthalmology and Otolaryngology

Chairman: Dr. Hilliard Wood, 700 Church St., Nashville, Tenn.
Secretary-Treasurer: Dr. H. C. Smith, Medical Arts Bldg., Nashville, Tenn.
Place: St. Thomas Hospital. Time: 8 p. m., third Monday of each month.

New Orleans Ophthalmological and Otolaryngological Society

President: Dr. Charles A. Bahn, 150 Baronne St., New Orleans.
Secretary-Treasurer: Dr. M. F. Meyer, Canal Bank Bldg., New Orleans.
Place: Eye, Ear, Nose and Throat Hospital. Time: Third Thursday of each month from October to June.

New York Academy of Medicine, Section of Ophthalmology

Chairman: Dr. Mark J. Schoenberg, 1160 Park Ave., New York.
Secretary: Dr. Algernon B. Reese, 73 E. Seventy-First St., New York.
Time: 8:30 p. m., third Monday of every month from October to May, inclusive.

Omaha and Council Bluffs Ophthalmological and Oto-Laryngological Society

President: Dr. William P. Haney, 107 S. Seventeenth St., Omaha.
Vice President: Dr. L. G. Howard, 532 First Ave., Council Bluffs, Iowa.
Secretary-Treasurer: Dr. W. A. Cassidy, 1620 Medical Arts Bldg., Omaha.
Place: University Club, 1914 Harney St., Omaha. Time: 6 p. m., dinner; 7 p. m., program; third Wednesday of each month from October to May.

Pittsburgh Ophthalmological Society

President: Dr. Edward B. Heckel, Jenkins Arcade Bldg., Pittsburgh.
Secretary: Dr. George H. Shuman, Park Bldg., Pittsburgh.
Place: Pittsburgh Academy of Medicine Bldg. Time: Fourth Monday of each month, except June, July, August and September.

Pittsburgh Slit-Lamp Society

President: Dr. W. W. Blair, 121 University Pl., Pittsburgh.
Secretary: Dr. George H. Shuman, Park Bldg., Pittsburgh.
Place: 121 University Pl. Time: Second Monday evening of every month, except June, July, August and September.

Richmond Ophthalmological and Oto-Laryngological Society

President: Dr. William R. Weisiger, Medical Arts Bldg., Richmond, Va.
Secretary: Dr. R. W. Vaughan, Medical Arts Bldg., Richmond, Va.
Place: Westmoreland Club. Time: 6 p. m., second Monday of each month from October to May.

Rochester Eye, Ear, Nose and Throat Society

Chairman: Dr. E. S. Amsler, 53 S. Fitzhugh St., Rochester, N. Y.
Secretary-Treasurer: Dr. E. W. O'Brien, 5 N. Goodman St., Rochester, N. Y.
Place: Rochester Medical Association, 113 Prince St. Time: 8 p. m., third Monday of each month from October to May.

St. Louis Ophthalmic Society

President: Dr. Julius H. Gross, Carleton Bldg., St. Louis.
Secretary: Dr. Carl T. Eber, 1006 Carleton Bldg., St. Louis.
Place: Oscar Johnson Institute. Time: Clinical meeting 5:30 p. m., dinner and scientific meeting 6:30 p. m., fourth Friday of each month from October to April, inclusive, except December.

San Antonio Ophthalmolo-Oto-Laryngological Society

President: Dr. Scott C. Applewhite, 705 E. Houston St., San Antonio, Texas.
Secretary-Treasurer: Dr. E. D. Dumas, 425 Medical Arts Bldg., San Antonio.
Place: Bexar County Medical Library. Time: 8 p. m., first Tuesday of each month from October to May.

San Francisco County Medical Society, Section on Eye, Ear, Nose and Throat

Chairman: Dr. Robert C. Martin, 384 Post St., San Francisco.
Secretary: Dr. George N. Hosford, 490 Post St., San Francisco.
Place: Society's Building, 2180 Washington St., San Francisco.
Time: Third Tuesday of every month except May, June, July and December.

Shreveport Eye, Ear, Nose and Throat Society

President: Dr. J. L. Scales, Medical Arts Bldg., Shreveport, La.
Secretary-Treasurer: Dr. W. L. Atkins, 505 Medical Bldg., Shreveport, La.
Place: 1240 Texas Ave. Time: 7:30 p. m., first Monday of each month from October to June.

Spokane Academy of Ophthalmology and Oto-Laryngology

President: Dr. David Hartin, Old National Bank Bldg., Spokane, Wash.
Secretary: Dr. W. W. Henderson, Paulsen Medical and Dental Bldg., Spokane, Wash.
Place: Paulsen Medical and Dental Library. Time: 8 p. m., fourth Tuesday of each month except June, July and August.

Syracuse Eye, Ear, Nose and Throat Society

President: Dr. M. G. Brown, 713 E. Genesee St., Syracuse, N. Y.
Secretary-Treasurer: Dr. A. Harry Rubenstein, Medical Arts Bldg., Syracuse, N. Y.
Place: University Club. Time: Second Friday of each month except June, July and August.

Tulsa Academy of Ophthalmology and Otolaryngology

President: Dr. Roy Dunlap, 108 W. Sixth St., Tulsa, Okla.
Secretary-Treasurer: Dr. Marvin D. Henley, 108 W. Sixth St., Tulsa, Okla.
Place: Medical Arts Bldg. Time: Monthly.